Vol. LXXXIX AUGUST, 1936 N. S. Vol. 42, No. 2

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of the
American Veterinary Medical Association

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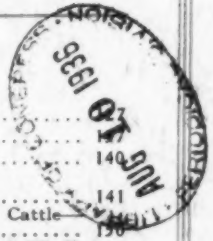

CONTENTS

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INFORMATION to the effect that the American Veterinary Medical Association will be invited to hold its seventy-fourth annual convention at Omaha in 1937 recalls the thirty-fifth annual meeting convened in that city, September 6, 1898, under the presidency of Dr. D. E. Salmon, distinguished American veterinarian and scientist, organizer and first chief of the United States Bureau of Animal Industry.

THE thirty-fifth annual meeting was comprised largely of pioneer graduates of the Middle West who had ventured to entertain the national association west of the Missouri River for the first time. The delegation made important association history:—

THE name of the Association was changed from "United States Veterinary Medical Association" to "American Veterinary Medical Association" in the spirit of expanding its jurisdiction to neighboring countries, and the Committee on Local Arrangements planned and conducted the Association's first surgical clinic, establishing a precedent that has contributed richly to the general interest in organized veterinary medicine.

THAT Omaha, the center of a vast live stock industry and of a large population of worthy, hard-working veterinarians in every branch of the service, will appreciate the honor of being host for the seventy-fourth annual convention of the A. V. M. A. is the foregone conclusion of

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H. Preston Hoskins, Secretary-Editor, 221 N. LaSalle St., Chicago, Ill.

Vol. LXXXIX, N. S. Vol. 42 AUGUST, 1936 No. 2

RECORD-BREAKING MEETING IN PROSPECT

All indications point to a record-breaking attendance at the Columbus convention this month. In the first place, the location of the meeting is favorable. Ohio has a large number of veterinarians. So have three of the adjoining states, Pennsylvania on the east, Michigan on the north, and Indiana on the west. In these four states there are about 2,300 veterinarians. Then, short distances away are New York, Illinois and Wisconsin, with about 2,250 more. The states to the south and southeast, including Kentucky, West Virginia, Maryland, Tennessee and the Carolinas, and the District of Columbia have approximately 700 veterinarians within their borders. These 13 states and the District of Columbia, with almost one-half of the veterinarians in the United States, will contribute 80 per cent of the attendance at Columbus.

Then the program. Advance comments are to the effect that it is one of the best ever arranged. The section officers certainly have done their best to make it such, with a total of 40 papers on almost as many subjects in the five sections. In the Section on Small Animals, a prominent member has been selected to open

the discussion on each paper. In the Section on Sanitary Science and Food Hygiene, meat inspection will be given a prominent place in the sun. In the Section on General Practice, all classes of large animals—horses, cattle, sheep and swine—will receive attention. In the Section on Research and the Section on Poultry, the results of numerous researches and investigations will be reported by our laboratory workers. Do not overlook the program for the general session on Wednesday morning, including addresses by two invited guests, Mr. Samuel R. Guard, editor of the *Breeder's Gazette*, and Mr. S. A. Postle, of the U. S. Food and Drug Administration.

Now, if the weather man will give Columbus a break the second week in August, what else could we wish?

REGISTRATION

Everybody attending the Columbus convention must register. This covers both members and non-members. Each person will pay a registration fee of $1.00 and will receive a badge, which will entitle the wearer to attend the various sessions and to participate in all free entertainment features which are put on by the Committee on Local Arrangements. The Ohio veterinarians are going to considerable expense to provide a most excellent program of entertainment for the ladies and children, and the cost of this entertainment is considerably more than the registration fee. It is very important that everybody register just as soon after arrival as possible. Tickets for the alumni dinners and general banquet should be secured just as early as possible, in order that the hotel may make the necessary arrangements for accommodating all who desire to attend these affairs.

SIR ARNOLD THEILER DIES

A press dispatch from London reports the death of Sir Arnold Theiler, on July 25, 1936, at the age of 68. He had been connected with the Imperial Bureau of Animal Health since 1933, except for a year (1934-35) spent in the Union of South Africa as Director of Veterinary Education and Research at Pretoria. Sir Arnold was in the United States in 1923 and again in 1934. On the first occasion he attended the A.V.M.A. meeting in Montreal, and was elected to honorary membership at that time. Following the meeting he visited many veterinary institutions

throughout the United States. In 1934, he attended the Twelfth International Veterinary Congress in New York.

CLINIC PROGRAM

One of the outstanding features of the Columbus meeting will be the clinic, which will occupy the entire day, Friday, August 14. The program indicates that the clinic will be the most comprehensive ever arranged for an A. V. M. A. convention. Dr. W. F. Guard, who will direct the clinic, has been hard at work for over six months planning the details. Assisting Dr. Guard are the chairmen of the six major sections of the clinic and a staff of well-known veterinarians assigned to each section. In addition to the surgical clinics for horses, cattle, sheep, swine, small animals and poultry, there will be a number of laboratory sections. An outline of the program for the clinic follows and it is hoped that it will be possible to carry out the entire program as arranged, although several operations and demonstrations necessarily are listed tentatively.

SECTION I—HORSES

Dr. Reuben Hilty, *Chairman*, Toledo, Ohio

OPERATIONS AND DEMONSTRATIONS

Sterility Demonstrations—
Dr. R. P. Marsteller, College Station, Tex.
Dr. A. A. Lenert, College Station, Tex.

Artificial Insemination in Mares—
Dr. Fred W. Miller, Beltsville, Md.
Major C. E. Cook, V. C., U. S. Army.

Umbilical Hernia Under General Anesthesia—
Dr. R. R. Dykstra, Manhattan, Kan.
Dr. J. P. Hutton, East Lansing, Mich.

Demonstration of Castration: Two Procedures—
Dr. J. P. Hutton, East Lansing, Mich.

Epidural Anesthesia and Its Indications—
Dr. R. R. Dykstra, Manhattan, Kan.

Miscellaneous Cases for Diagnosis—
Clinic Staff.

Section II—Cattle

Dr. C. H. Case, *Chairman*, Akron, Ohio

Operations and Demonstrations

Mastitis: Routine Methods of Diagnosis and Methods of Prevention—
Dr. D. H. Udall, Ithaca, N. Y.
Dr. S. D. Johnson, Ithaca, N. Y.
Dr. C. H. Case, Akron, Ohio.
Dr. Joseph Drayer, Columbus, Ohio.

Various Methods of Applying the Test for Bang's Disease—
Dr. E. W. Roberts, Reynoldsburg, Ohio.

Chemical Determinations as Applied to Blood, Urine and Milk, Having Diagnostic and Clinical Significance—
Dr. C. E. Hayden, Ithaca, N. Y.
Dr. R. E. Nichols, Columbus, Ohio.
Dr. Jesse Sampson, Ithaca, N. Y.

Teat Operations—
Dr. T. H. Ferguson, Lake Geneva, Wis.

Ovariectomy—
Dr. E. R. Frank, Manhattan, Kan.

Dehorning—
Dr. M. A. Emmerson, Philadelphia, Pa.

Obstetrical Demonstrations—
Dr. M. G. Fincher, Ithaca, N. Y.

Exhibit of Study of Physiology of the Rumen of Steers—
Dr. A. F. Schalk, Columbus, Ohio.

Section III—Swine

Dr. B. H. Edgington, *Chairman*, Columbus, Ohio

Operations and Demonstrations

Cesarean Operation—
Dr. George Lies, Fort Recovery, Ohio.

Castration of Aged Boars—
Dr. John B. Bryant, Mount Vernon, Iowa.

Castration of Pigs—
Dr. E. A. Downs, Mount Sterling, Ohio.

Rectal Prolapse in Swine—
Dr. E. J. Starbuck, Port William, Ohio.

Umbilical Hernia in Swine—
Dr. D. D. Baker, Wabash, Ind.

Cryptorchid Operation—
Dr. E. V. Hover, Lima, Ohio.
Nutritional Anemia in Pigs—
Dr. H. C. H. Kernkamp, Saint Paul, Minn.
Use of Stomach-Tube in Swine Practice—
Dr. C. W. Fogle, Leipsic, Ohio.
Epidural Anesthesia in Swine—
Dr. E. R. Frank, Manhattan, Kan.
Methods of Restraint in Swine—
Dr. D. D. Baker, Wabash, Ind.
Demonstration of Mange in Swine—
Dr. J. B. Hollenbeck, Columbus, Ohio.

Section IV—Sheep

Dr. P. T. Engard, *Chairman*, Marysville, Ohio

Operations and Demonstrations

Contagious Ecthyma (Sore Mouth) in Lambs—
Dr. A. F. Schalk, Columbus, Ohio.
Dr. I. B. Boughton, Sonora, Tex.
Diagnosis of Scab. Demonstration of Lice—
Dr. P. T. Gillie, Columbus, Ohio.
Dr. R. S. Smiley, Cromers, Ohio.
Dipping and Equipment—
Dr. H. R. Hinchman, Canal Winchester, Ohio.
Dr. A. E. Fogle, Worthington, Ohio.
Intestinal Parasites—
Dr. E. D. Martin, Reynoldsburg, Ohio.
Foot Rot and Treatment—
Dr. P. T. Gillie, Columbus, Ohio.
Herding Sheep with Dogs—
Mr. L. F. Taylor, Marysville, Ohio.
Administration of Drugs—
Dr. P. T. Engard, Marysville, Ohio.

Section V—Small Animals

Dr. D. W. Ashcraft, *Chairman*, Columbus, Ohio

Operations and Demonstrations

Amputation of the Breasts in Dogs—
Dr. C. F. Schlotthauer, Rochester, Minn.
General Presentation of Laboratory Aids to Diagnosis—
Dr. M. L. Morris, New Brunswick, N. J.

Skin Diseases—
Dr. J. V. Lacroix, Evanston, Ill.

Cesarean Section—
Dr. J. A. Campbell, Toronto, Canada.

Examination, Diagnosis, Reduction and Fixation for the Coxo-Femoral Dislocation and Fracture of the Femoral Neck in the Dog—
Dr. E. F. Schroeder, Boston, Mass.

Tonsillectomy—
Dr. D. A. Eastman, Moline, Ill.

Operation for Relief of Otorrhea—
Dr. J. V. Lacroix, Evanston, Ill.

Demonstration of the Use of a Special Instrument for Removal of Foreign Bodies from the Stomach—
Dr. W. A. Young, Chicago, Ill.

Cecal Injections—
Dr. H. N. Corenzwit, Philadelphia, Pa.

Euthanasia in Dogs—
Dr. H. N. Corenzwit, Philadelphia, Pa.

SECTION VI—POULTRY

Dr. J. T. Burriss, *Chairman*, Reynoldsburg, Ohio

OPERATIONS AND DEMONSTRATIONS

Pullorum Disease—

1. Technic for collecting blood samples:
   A. Syringe method.
   B. Nicking method.
   Demonstrator: Dr. P. H. Seitz, Harrisburg, Pa.
2. Standard tube test method.
   Demonstrator: Dr. E. N. Moore, Charleston, W. Va.
3. Rapid serum test method.
   Demonstrator: Dr. A. W. Deem, Columbus, Ohio.
4. Rapid whole-blood, stained-antigen test method.
   Demonstrator: Dr. H. J. Stafseth, East Lansing, Mich.
5. Pathological and bacteriological display.
   Demonstrator: Dr. C. T. Mingle, Columbus, Ohio.

Fowl-Pox—

1. Technic of vaccination:
   A. Stick method.
   B. Feather follicle method.

2. Specimens:
   A. Live vaccinated birds in reactionary period:
      I. Chicken strain of virus.
      II. Pigeon strain of virus.
   B. Infected birds.
   C. Immune birds.

   Demonstrator: Dr. E. L. Brunett, Ithaca, N. Y.

Laryngotracheitis (Infectious Bronchitis)—

1. Technic of vaccination.
2. Vaccinated birds in reactionary period.
3. Infected birds.
4. Technic for the propagation of viruses upon the chorioallantoic membrane of the hen's egg.

   Demonstrator: Dr. C. A. Brandly, Urbana, Ill.

Avian Tuberculosis—

1. Technic for tuberculin testing.
2. Injected birds in reactionary period.
3. Infected birds.

   Demonstrator: Dr. E. S. Augsburger, Columbus, Ohio.

Fowl Paralysis—

1. Technic for general diagnosis, based on pathologic tissues.
2. Specimens:
   A. Affected birds.
   B. Prepared microscopic slides of pathologic tissues.

   Demonstrator: Dr. E. P. Johnson, Blacksburg, Va.

Fungous Diseases—

1. Technic of autopsy to demonstrate the common anatomical locations of these type infections.
2. Specimens:
   A. Prepared lantern-slides showing the infective agents and pathologic tissues.
   B. Live cultures of different fungi.

   Demonstrator: Dr. Erwin Jungherr, Storrs, Conn.

Coccidiosis and Enterohepatitis (Blackhead)—

1. Technic for general diagnosis.
2. Specimens:
   A. Live affected specimens.
   B. Prepared pathologic tissue.

   Demonstrator: Dr. J. P. Delaplane, Kingston, R. I.

Parasite Display—

1. Prepared specimens of different external and internal parasites associated with parasitic diseases of poultry.

2. Technic for general identification.
   Demonstrators: Dr. R. E. Rebrassier, Columbus, Ohio.
   Dr. C. A. Woodhouse, Columbus, Ohio.

Pathology Display—

1. Prepared specimens of pathologic tissues of various diseases affecting poultry.
   Demonstrator: Dr. L. W. Goss, Columbus, Ohio.

One Form of Technic for Routine Autopsy—
   Demonstrator: Dr. P. C. Bennett, Reynoldsburg, Ohio.

Technic for Confining Live Birds—

1. Flock confinement.
2. Individual handling of birds.
3. Culling for standard breed disqualifications.
4. Leg-banding for individual bird identification.
   Demonstrator: Dr. F. A. Young, Delphos, Ohio.

SECTION VII—SMALL-ANIMAL LABORATORY

Dr. M. L. Morris, *Chairman*, New Brunswick, N. J.

Dr. A. R. Theobald and Mr. Adler, Technician, Cincinnati, Ohio

1. Standardizing Conditions Prior to the Collection of Samples.
2. The Collection of Samples for Clinical Laboratory Determinations:
   A. Care, preservation and handling.
3. Examination of Specimens:
   A. Feces:
      a. Macroscopic.
      b. Chemical.
      c. Microscopic.
      d. Bacteriological.
   B. Skin:
      a. Macroscopic.
      b. Microscopic.
      c. Bacteriological.
      d. Special methods.
   C. Urine:
      a. Macroscopic.
      b. Chemical.
      c. Microscopic.
      d. Bacteriological.
   D. Blood:
      a. Hematology:
         (1) Erythrocyte and leukocyte counts.
         (2) Hemoglobin determinations.

(3) Blood parasites:
I. Filaria (3 or more methods).
II. Piroplasma.
(4) Sedimentation rate.

b. Chemistry:
(1) Explanations only. No determinations.

c. Serology:
(1) Primary methods of clinical value.

d. Bacteriology.

4. Special Demonstrations of Clinical Importance:
A. The Use of Violet Ray in Diagnosis of Skin Lesions (Wood's filter).
B. Lantern Slides.
C. Interesting and Unusual Specimens (Microscopic slides).

SECTION VIII—LABORATORY SECTION

Dr. C. E. Hayden, *Chairman*, Ithaca, N. Y.
Dr. R. E. Nichols, Columbus, Ohio
Dr. Jesse Sampson, Ithaca, N. Y.

Chemical Determinations as Applied to Blood, Urine and Milk, Having Diagnostic and Clinical Significance.

A. Demonstration of equipment and methods for the determination of blood constituents having greatest clinical significance.
B. Demonstration of equipment and methods for the determination of urine constituents having most clinical significance.
C. Rapid chemical tests for the detection of mastitis. A test for the detection of acetone bodies in the milk in severe cases of ketosis or acetonemia.

SECTION IX—LABORATORY ON PATHOLOGY

Dr. Leonard W. Goss, *Chairman*, Columbus, Ohio
Dr. George D. Jelen, Columbus, Ohio
Dr. R. P. Wagers, Columbus, Ohio

Fresh and Permanent Mounted Specimens.

SECTION X—LABORATORY ON PARASITOLOGY

Dr. R. E. Rebrassier, *Chairman*, Columbus, Ohio
Dr. C. A. Woodhouse, Columbus, Ohio

Parasite Exhibit.

SECTION XI—DEMONSTRATION OF AGGLUTINATION TESTS FOR BANG'S DISEASE

Dr. E. W. Roberts, *Chairman*, Reynoldsburg, Ohio

A. Clear Serum Test:

1. Standard Tube Test.
2. Plate or Rapid Method.
   Dr. E. W. Roberts, Reynoldsburg, Ohio.

B. Whole-Blood Stained-Antigen Method.
   Dr. T. T. Genre, U. S. Bureau of Animal Industry.

SECTION XII—ALL-DAY CLINICAL INSTRUCTION ON MASTITIS

Dr. D. H. Udall, *Chairman*, Ithaca, N. Y.
Dr. S. D. Johnson, Ithaca, N. Y.

EXHIBITORS AT COLUMBUS

ABBOTT LABORATORIES, North Chicago, Ill.
Pharmaceuticals.

ALLIED LABORATORIES, Kansas City, Mo.
Biologicals and pharmaceuticals.

BECTON, DICKINSON AND CO., Rutherford, N. J.
Syringes, thermometers.

CAMERON SURGICAL SPECIALTY CO., Chicago, Ill.
Surgical specialties.

COLUMBUS SERUM CO., Columbus, Ohio.
Biologicals and pharmaceuticals.

CORN STATES SERUM CO., Omaha, Neb.
Anti-hog cholera serum.

FAICHNEY INSTRUMENT CORPORATION, Watertown, N. Y.
Instruments.

FORT DODGE SERUM CO., Fort Dodge, Iowa.
Biologicals and pharmaceuticals.

GENERAL ELECTRIC X-RAY CORPORATION, Chicago, Ill.
X-ray equipment.

HAVER-GLOVER LABORATORIES, Kansas City, Mo.
Biologicals and pharmaceuticals.

JENSEN-SALSBERY LABORATORIES, INC., Kansas City, Mo.
Biologicals and pharmaceuticals.

JUDY PUBLISHING CO., Chicago, Ill.
Dog publications.

LEDERLE LABORATORIES, INC., New York, N. Y.
Biologicals and pharmaceuticals.

ASHE LOCKHART, INC., Kansas City, Mo.
Biologicals.

MERCK AND COMPANY, Rahway, N. J.
Pharmaceuticals.

NATIONAL LABORATORIES, Kansas City, Mo.
Biologicals and pharmaceuticals.
NORDEN LABORATORIES, Lincoln, Neb.
Biologicals and pharmaceuticals.
SPICER AND COMPANY, Glendale, Calif.
Pharmaceuticals.
R. J. STRASENBURGH CO., Rochester, N. Y.
Pharmaceuticals.
SWIFT AND COMPANY, Chicago, Ill.
"Pard" dog food.
VITAMIN PRODUCTS CO., Milwaukee, Wis.
Vitamin products.
WILSON AND CO., INC., Chicago, Ill.
"Ideal" dog food.

IDAHO AFFILIATES

At a meeting of the Idaho Veterinary Medical Association held at Twin Falls on June 22-23, it was decided to affiliate with the American Veterinary Medical Association. The Idaho organization has not been holding regular meetings during recent years and for that reason action on affiliation had been deferred. With Idaho in the fold, we now have every one of the 48 state associations in the country affiliated with the A.V.M.A.

APPLICATIONS FOR MEMBERSHIP

(See July, 1936, JOURNAL)

FIRST LISTING

BAILEY, LEE K. 123 S. Randolph, Lexington, Va.
D. V. M., Iowa State College, 1933
Vouchers: I. D. Wilson and L. E. Starr.

BEARD, DONALD C. Box 994, Sanford, N. C.
D. V. M., Colorado State College, 1935
Vouchers: Wm. Moore and W. A. Hornaday.

BERRY, THOMAS A. 2101 Milvia St., Berkeley, Calif.
D. V. M., Ohio State University, 1933
Vouchers: Harold H. Groth and Maurice L. Boevers.

DAUGHTREY, F. D. 355 E. Fifth Ave., Lancaster, Ohio
D. V. M., Ohio State University, 1929
Vouchers: A. J. DeFosset and Jay W. Reeder.

DAVIDSON, L. N. 905 W. Loucks St., Sheridan, Wyo.
D. V. M., Saint Joseph Veterinary College, 1922
Vouchers: H. D. Port and W. A. Sullivan.

DONAHUE, MICHAEL J. 136 W. 53rd St., New York, N. Y.
D. V. M., Cornell University, 1935
Vouchers: C. P. Zepp and Cassius Way.

ELLIOTT, BERTON J. Albany, Ohio
D. V. M., Ohio State University, 1936
Vouchers: Walter R. Krill and C. R. Donham.

ELLIS, CHARLES H., JR. 624 Union St., Brunswick, Ga.
D. V. M., Indiana Veterinary College, 1921
Vouchers: J. C. Flynn and J. E. Severin.

HENDRICKS, STANLEY L. 210 Cotton States Bldg., Nashville, Tenn.
D. V. M., Iowa State College, 1934
Vouchers: Hugh L. Fry and E. B. Parker.

HOLMES, JOHN M. 3750 Carnegie Ave., Cleveland, Ohio
D. V. M., Ohio State University, 1932
Vouchers: N. L. Siplock and Solon Gillen.

INGMAND, EUGENE B. Box 815, Zionsville, Ind.
B. S., Iowa State College, 1933
D. V. M., Iowa State College, 1934
Vouchers: S. H. Regenos and J. L. Kixmiller.

JOSSE, CHARLES K. Germantown, Ohio
D. V. M., Ohio State University, 1936
Vouchers: Walter R. Krill and W. F. Guard.

KERN, WILLIAM H. Box 735, Winston-Salem, N. C.
D. V. M., Kansas City Veterinary College, 1913
Vouchers: J. H. Brown and A. A. Husman.

KNUDSON, ROBERT L. State House, Augusta, Me.
D. V. M., Ohio State University, 1934
Vouchers: W. C. Dendinger and J. F. Witter.

LEE, WALTER H. Big Piney, Wyo.
D. V. M., San Francisco Veterinary College, 1913
Vouchers: H. D. Port and W. A. Sullivan.

LINDLEY, WILLIAM H. Lawrenceburg, Ind.
D. V. M., Kansas State College, 1933
Vouchers: Walter K. York and C. C. Donelson.

MCQUOWN, JOHN B. 2300 E. Broadway, Tucson, Ariz.
D. V. M., Ohio State University, 1919
Vouchers: Calvert T. Guilfoyle and Ward R. Lee.

MUSKOVIN, ALBIN 61 W. Sunrise Highway, Merrick, N. Y.
D. V. M., Cornell University, 1919
Vouchers: James R. Kinney and C. P. Zepp.

NOONAN, CYRIL J. Mamaroneck Ave. at Saxonwood Rd., White Plains, N. Y.
D. V. M., Cornell University, 1928
Vouchers: Cassius Way and C. P. Zepp.

PHARR, ROY R. 35 N. Heber St., Beckley, W. Va.
D. V. M., Kansas City Veterinary College, 1917
Vouchers: S. E. Hershey and H. M. Newton.

PRITCHARD, JOHN W. Lena, Wis.
M. D. V., McKillip Veterinary College, 1910
Vouchers: James S. Healy and W. R. Winner.

SPIERLING, WILLIAM E. New Post Office Bldg., Columbus, Ohio
D. V. M., Chicago Veterinary College, 1916
Vouchers: Geo. D. Jelen and T. E. Nichols.

STARK, HERMAN 397 Carlton Ave., Brooklyn, N. Y.
D. V. S., New York-American Veterinary College, 1903
Vouchers: Ray W. Gannett and Christian G. Rohrer.

STATON, BRUCE H. N. C. Department of Agriculture, Raleigh, N. C.
B. S., North Carolina State College, 1932
D. V. M., Alabama Polytechnic Institute, 1934
Vouchers: J. H. Brown and L. J. Faulhaber.

WATSON, CLEMENT E. Hotel Lodi, Lodi, Calif.
D. V. M., Kansas State College, 1935
Vouchers: H. J. Hearrington and D. E. Settle.

WERNICOFF, NATHAN 105-06 Metropolitan Ave., Forest Hills, N. Y.
D. V. M., Cornell University, 1931
Vouchers: Arthur D. Goldhaft and R. S. MacKellar, Jr.

WHITLOCK, JOHN H. Kansas State College, Manhattan, Kan.
D. V. M., Iowa State College, 1934
M. Sc., Kansas State College, 1935
Vouchers: E. E. Leasure and R. R. Dykstra.

WILSON, WELLS M. Williamsport, Ohio
D. V. M., Ohio State University, 1936
Vouchers: W. F. Guard and Leonard W. Goss.

Applications Pending

SECOND LISTING

(See July, 1936, JOURNAL)

Arron, Daniel P., 312 32nd Ave., Seattle, Wash.
Baum, Harvey, 24 Maple St. S. E., Massillon, Ohio.
Berger, George, 18 Edmondson Ave., Lexington, Va.
Berliner, Meyer, 6 Margot Pl., Great Neck, L. I., N. Y.
Bordner, Lawson A., 903 13th Ave. S., Nampa, Idaho.
Bradley, Donald H., 1569 N. High St., Columbus, Ohio.
Brand, Rudolph W., 613 Lincoln St., Longmont, Colo.
Cantrall, Emmett W., Likely, Calif.
Christopher, Burton C., 303 E. Union Ave., Olympia, Wash.
Collins, Floyd M., Lyons, Neb.
Cook, Roger A., 205 N. Sprague Ave., Bellevue, Pittsburgh, Pa.
Cox, Danford L., Box 313, Wausau, Wis.
Cromley, Curtis W., Ashville, Ohio.
Dodge, Ida Mae, Harrison Ave. at Three-Mile, Butte, Mont.
Dodge, Roger E., Harrison Ave. at Three-Mile, Butte, Mont.
Dougherty, Robert W., Goochland, Va.
Fleming, John, Box 313, Wausau, Wis.
Geyer, Harry G., Grove City, Ohio.
Green, Kenneth L., R. 4, Golden, Colo.
Guard, William P., Friday Harbor, Wash.
Hackett, Clarence P., Kinsman, Ohio.
Hammond, William H., R. 1, Monterey, Calif.
Henkel, Ernest L., 1606 Ruby St., Pullman, Wash.
Herman, Leslie F., 37 W. Bagley Rd., Berea, Ohio.
Holmberg, Gerald W., Sanbornville, N. H.
Houk, William S., 402 Park Dr., Muscatine, Iowa.
Jones, Kenneth S., Kenton, Ohio.
Joneschild, William R., 616 S. Adams, Tacoma, Wash.
Jurden, Richard H., Box 3002, San Carlos, Calif.
Koutz, Fleetwood R., 204 Spaulding St., Pullman, Wash.
Leggett, William R., 99 Windsor St., Waterbury, Conn.
Long, Gerald H., Mount Sterling, Ill.
Miller, James J., 1371 Fulton St., San Francisco, Calif.
Mills, John W., Ohio State University, Columbus, Ohio.
Moore, Morris E., Flora, Ind.
Motteler, George F., Colville, Wash.
Nixon, Gaylord J., R. 5, Mansfield, Ohio.
Palmer, Charles A., 610 W. Masonic St., Gainesville, Fla.
Rice, Allen W., 507 Federal Bldg., Little Rock, Ark.
Spangler, Harold M., 1116 Bluemont Ave., Manhattan, Kan.
Stephan, Sol G., 30 Erkenbrecher Ave., Cincinnati, Ohio.
Talcott, Robert V. P., 925 California Dr., Burlingame, Calif.

Twisselmann, Norman M., Maricopa, Calif.
Utterback, John A., Jr., Milner, Colo.
Wagers, Robert P., Ohio State University, Columbus, Ohio.
Weigand, Harry G., 2750 S. Broadway, Englewood, Colo.
Williams, George A., 831 W. Milford St., Glendale, Calif.
Winiecki, Henry F., 702 Cherry St., Raymond, Wash.

The amount which should accompany an application filed this month is $7.08, which covers membership fee and dues to January 1, 1937, including subscription to the JOURNAL.

COMING VETERINARY MEETINGS

Connecticut Veterinary Medical Association. New London, Conn. August 5, 1936. Dr. Geo. E. Corwin, Secretary, State Office Bldg., Hartford, Conn.

Houston Veterinary Association. Houston, Texas. August 6, 1936. Dr. D. B. Strickler, Secretary, 317 Federal Bldg., Houston, Texas.

San Diego County Veterinary Medical Association. San Diego, Calif. August 11, 1936. Dr. Donald E. Stover, Secretary, Zoölogical Research Bldg., Balboa Park, San Diego, Calif.

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INTERNAL HYDROCEPHALUS IN DOGS*

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Hydrocephalus is a disease characterized by an increased quantity of cerebro-spinal fluid within the cranium, and it causes increased intracranial pressure and compression of the brain. The excess fluid may be within the ventricles of the brain, constituting internal hydrocephalus, or it may be in the subdural space, constituting external hydrocephalus. An associated dilatation of the ventricles, however, is not uncommon with the latter type of hydrocephalus. This paper will deal chiefly with the ventricular or internal type of hydrocephalus and its occurrence in dogs will be discussed.

According to the available literature, internal hydrocephalus is not infrequent in man and lower animals; but reports of cases in which it has occurred in lower animals are meager. Huyra and Marek[1] stated that it is most frequently observed in horses, and is then a common cause of "sleepy staggers." However, data as to the frequency of its occurrence are not available. They mentioned that Fröhner observed 20 cases of internal hydrocephalus in 70,000 dogs, an incidence of one in every 3,500 dogs examined. They further stated that Roquet observed this disease in one dog and Schindelka noted it in a swine. Milks,[2] in 1918, reported the occurrence of hydrocephalus in two kittens of a litter of six. Cholesteatomas were found in two other kittens of the same litter. Serrano,[3] in 1933, reported one case of internal hydrocephalus in a dog, and I,[4] in 1934, reported two similar cases.

Since my previous report[4] of internal hydrocephalus in dogs, I have observed the disease in five additional dogs. The clinical history and findings at necropsy in these dogs will be discussed in this paper.

The etiology of internal hydrocephalus is varied. It may be congenital or acquired, acute or chronic. Church and Peterson[5] stated that some families appear to have a hereditary tendency toward it. They mentioned, however, that congenital or acquired obstruction of the foramina or aqueduct is the common cause of internal hydrocephalus. McKinley,[6] in Bell's "Text Book of Pathology," stated that tumors, inflammatory adhesions and congenital atresia are the most frequent causes of obstruction of

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the foramina and aqueduct in man, although in some instances, in infants, no cause for the hydrocephalus can be demonstrated.

Wiggers[7] stated that the choroid plexuses are the chief source of the cerebrospinal fluid. It leaves the ventricular spaces through the foramina and aqueduct, passing out into the subarachnoid space where it seeps downward and upward and ultimately passes directly into the veins through the arachnoid villi. Only a small portion escapes along the nerve sheaths and vessels. He stated that hydrocephalus can be produced experimentally by ligating the great vein of Galen which drains the choroid plexuses. It is possible that obstruction of this vessel may occur and cause hydrocephalus clinically but this is not a frequent occurrence. He further mentioned that although obstruction of the normal circulation of cerebrospinal fluid is most likely to occur in the narrow portions of the ventricles, foramina and aqueduct, it may also occur in the narrow subarachnoid space at the level of the tentorium. Dandy[8] produced obstruction in this region experimentally in dogs. Obstruction at this point produces a communicating type of hydrocephalus, that is, the foramina and aqueduct remain patent. Some cases of so-called idiopathic internal hydrocephalus may be of this type.

Hutyra and Marek[1] stated that, in lower animals, acquired internal hydrocephalus occurs most frequently following acute meningo-encephalitis, acute encephalitis, frequent protracted hyperemia, and tumors. However, since true intracranial neoplasms are extremely infrequent in lower animals, these investigators undoubtedly are referring to cholesteatoma, a rather frequent tumor in horses. They mentioned that parasitic cysts of Echinococcus and Coenurus are uncommon causes of obstruction of the foramina and aqueduct.

A hereditary predisposition toward hydrocephalus was noted by Dexler.[9] His investigations tended to show that its frequent occurrence in horses is attributable to anatomic conditions of the cranium.

The symptoms of internal hydrocephalus may be indefinite and confusing. They vary with age and the degree of hydrocephalus present. If it develops before the fontanels and cranial sutures are closed, the skull may be greatly enlarged and diagnosis can be made from this observation alone. In such cases the ventricles may be so dilated that the brain is little more than a thin membrane, and cerebral injury in these cases is severe. McKinley stated that children so affected have various grades of feeble-mindedness and paralysis. However, mild grades of hydrocephalus may not cause evidence of mental or physical impairment.

Acquired internal hydrocephalus in adults cannot cause enlargement of the skull because the fontanels and cranial sutures are closed and firmly bound together. A marked increase of cerebrospinal fluid may therefore cause grave symptoms of injury to the brain not unlike those of brain tumor. The symptoms manifested in man are headache, nausea, vomiting, vertigo, Cheyne-Stokes respiration, various mental states, choked disks and amaurosis. Church and Peterson[5] called attention to the frequency of hemiplegia and impairment of memory. They also mentioned that convulsions and rigidity may be observed. The pupils are dilated and stationary and strabismus is not uncommon.

It is highly probable that lower animals experience the same symptoms as man, but recognition of some symptoms is difficult. Animals, of course, cannot tell us when they have headaches, nausea, or slight impairments of vision or hearing, nor can they explain their various mental states. One must rely entirely on the visible symptoms and behavior. Hutyra and Marek[1] stated that animals affected with internal hydrocephalus have a fixed, meaningless expression; they appear dumb, and if left undisturbed may remain in one place for a long time. In the early stages of the disease, horses may experience difficulty in stepping over objects or in turning suddenly. Affected animals when eating may forget to masticate and swallow their food, and permit it to drop from the mouth. The skin reflexes usually are diminished or abolished, though occasionally they are exaggerated. The heart action and respiration may be slowed. Fröhner[10] observed dullness, loss of smell, amaurosis, deafness, and uncertain gait in dogs affected with internal hydrocephalus. In the two cases of internal hydrocephalus in dogs previously reported by me,[4] changed expression and behavior, loss of memory and convulsions were prominent symptoms. Ataxia was noted in one case and choked disks and impaired vision in the other. The sense of balance of both dogs was easily disturbed. The symptoms observed in the five cases which I am reporting in this paper will be discussed in detail. Although the syndrome in each case indicated disease of the central nervous system, the cases were somewhat dissimilar.

Reports of Cases

Case 1: A male bull dog, eight weeks of age, manifested symptoms of disease of the central nervous system, especially of the spinal cord, when only six weeks of age, and at that time weakness of the hind legs and fecal and urinary incontinence were noted. The owner stated that these symptoms were progressive.

He also mentioned that similar symptoms had been observed in four other dogs of this breed, three males and one female. The males all died when quite young, but the female was still alive and was more than a year old and was pregnant. She, however, had lost the use of her hind legs. He wondered if the disease might be hereditary because it appeared four times among the offspring of a single male.

Physical examination revealed numerous small cutaneous ulcers on the anus, scrotum, prepuce and pads of both hind feet. These appeared to incite no tissue reaction. The dog's hind legs appeared to be weak and unsteady. Areas of anesthesia were noted

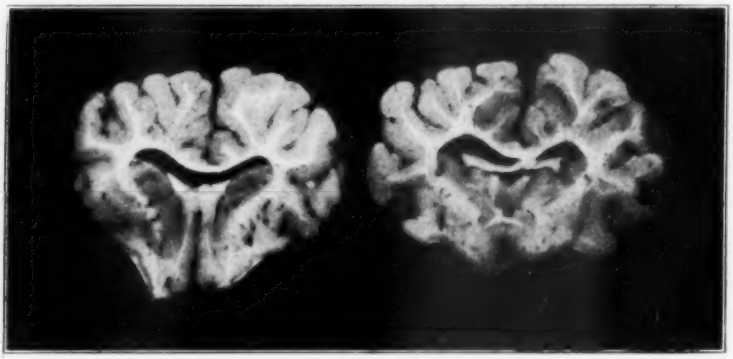

FIG. 1. Case 1. Cross section of brain. Note bilateral dilatation of the lateral ventricles.

in both hind legs and in the perineal region. His sight was apparently normal but his hearing was impaired. If left undisturbed he would lie quietly in one place heedless of speech and other noises, but if one touched him he would become very active. There was no spasticity of his legs, trunk or neck and his reflexes were quite normal. His temperature and pulse and respiration rates were normal. He had a voracious appetite and appeared well nourished. There was no gross deformity of his skull and vertebral column. *Spina bifida occulta* and intermedullary tumor of the spinal cord were considered, but diagnosis was withheld and the dog was placed under observation. All symptoms progressed rapidly and he died quite suddenly when exactly two months of age.

Necropsy revealed bilateral, internal hydrocephalus (fig. 1). There also was definite hydromyelia and the central canal of the spinal cord was open at its distal extremity (fig. 2). A condi-

tion similar to, but not typical of, *spina bifida occulta* was present. The cause of this apparently was congenital.

Case 2: A male fox terrier, three months of age, was brought to me because of weakness or posterior paralysis of two weeks duration. The owner stated that this dog had had no previous illness. It was thought that he had suffered an injury from having slipped on a recently waxed kitchen floor.

Physical examination revealed marked weakness of the hind legs and function was definitely impaired. There was no incontinence and no spasticity or rigidity of the legs. The reflexes were essentially normal. The dog manifested no pain in his spine when it was palpated, though he whined as if worried or

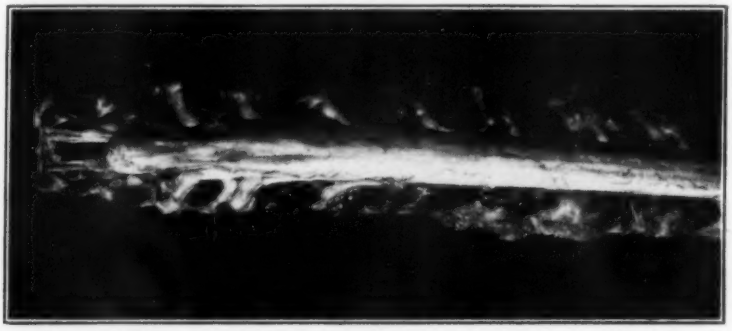

FIG. 2. Case 1. Note the open central canal in the lumbar portion of the spinal cord.

uncomfortable. Examination of his eyes with an ophthalmoscope revealed small hemorrhagic areas in the retina and some edema of the optic papilla in the right eye. This caused some slight impairment of vision. His temperature, sense of hearing, and pulse and respiration rates were essentially normal. There was no gross deformity of the skull and vertebral column. His appetite was good and he appeared to be well nourished.

The progressive nature of the disease and the general symptoms manifested were suggestive of an extradural tumor of the spinal cord. However, typical symptoms of pain in the spinal column and rigidity of the back and legs were absent. In view of the lesions in the right eye and the similar symptoms in the previous case, the possibility of internal hydrocephalus was considered, but because of the seemingly hopeless outlook for this dog he was destroyed.

Necropsy revealed marked dilation of the left lateral ventricle, confirming the tentative diagnosis of internal hydrocephalus (fig.

3). The cause of the obstruction in the lateral ventricle was not determined. There were no other demonstrable lesions.

Case 3: A male, white collie, four years or more of age, was not known to have had any previous severe illness. He had always stayed at home and had been a good pet. Two weeks before he was brought to me, he had begun wandering away from home and had become easily lost. If he walked into a place where he had to turn around to get out, he appeared unable to comprehend his situation and to escape from it. Once he waded into a pond

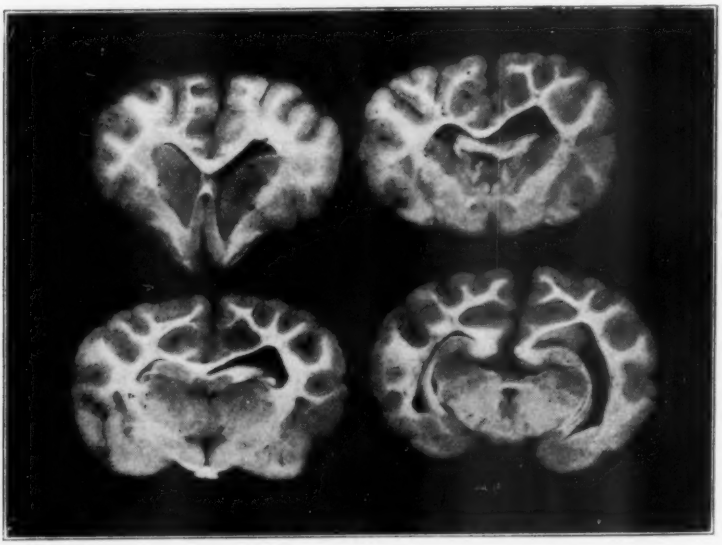

FIG. 3. Case 2. Note dilatation of the left lateral ventricle.

and remained there until he was forcibly removed. He occasionally would lie on his back and howl as if he suffered pain. His appetite was impaired, but he was very fat.

Physical examination did not reveal any deformity of this dog's skull or vertebral column. He manifested some weakness in his hind legs, but there was no definite paralysis. He had a dull, dumb, meaningless expression and his sight and sense of hearing were definitely impaired. Examination of his eyes revealed bilateral choked disks. His temperature was slightly elevated and his pulse and respiration rates were irregular. A tentative diagnosis of brain tumor, interal hydrocephalus or encephalitis was advanced and he was hospitalized for observation. His symptoms became progressively more severe and he was destroyed after three days.

Necropsy revealed moderate but well-marked dilation of both lateral ventricles. The cause of this was not determined. Other lesions could not be demonstrated.

Case 4: A male, mongrel, white bull terrier, four years of age, was raised in a kennel in which distemper was rather prevalent, but there was no record of his having had it. His owner stated that during the previous eight months or longer he had found it impossible to train or teach this dog to do things that any normal dog should do; and, too, it was noted that this dog muti-

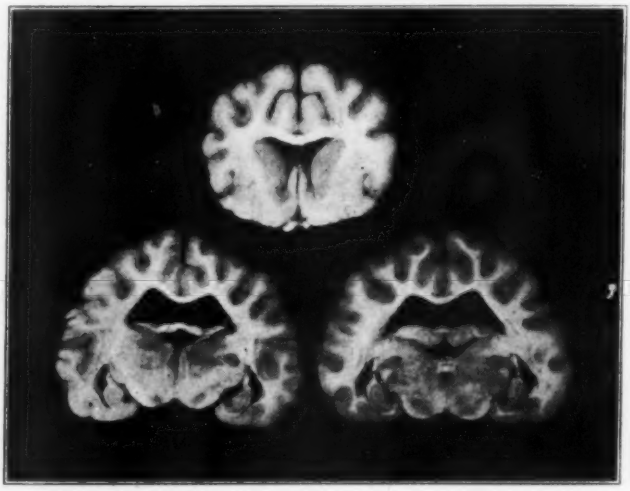

FIG. 4. Case 4. Marked bilateral internal hydrocephalus. Note that the fornix and the septum pellucidum are flattened.

lated his tail by chewing it. He had recently manifested some weakness in his hind legs and developed a number of cutaneous ulcers on his hind feet and a large sloughing wound on each thigh. He was thin and emaciated. Because of his deplorable condition, he was destroyed and brought to me for examination.

Necropsy revealed marked internal hydrocephalus (fig. 4). All ventricular spaces, the aqueduct, and the central canal were dilated. Grossly it appeared that the hydrocephalus in this case was due to obstruction of the foramina of Luschka and Magendie, or of the narrow arachnoid space at the level of the tentorium. The etiology of the obstructing lesion was not determined.

Case 5: A male Boston terrier, ten years of age, had a rather prolonged history of disease of the central nervous system. He was observed to have many convulsions over a period of eight

years. I procured him when he was two years of age. He experienced a sudden, unaccounted for convulsion the day he was delivered to me. Similar convulsions occurred each time he was subjected to any unusual treatment or change in environment, such as a ride in a car, visitors in our home, the packing of a bag preparatory to a trip and a host of other things. During these convulsive attacks he would fall to the floor and lie there for a minute or two, after which he would arise and run about quite unconscious of his surroundings for five minutes or longer.

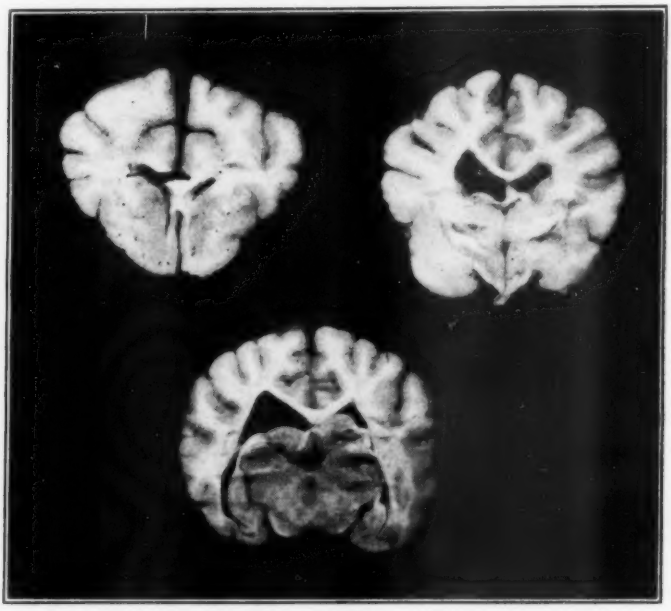

FIG. 5. Case 5. Note dilatation of the left lateral ventricle. The fornix is flattened and distorted on the affected side.

On a few occasions, when this occurred outside, he had run three to four city blocks. He was easily lost and, normally, never ventured out of sight of home. He was otherwise intelligent and had a good memory for things about our home.

The disease could not be controlled by dietary measures or medication and his condition appeared quite hopeless. A friend of ours thought the dog would outgrow this tendency for convulsions and agreed to take him. He kept him for five years, during which time the convulsions recurred each time the dog was subjected to any unusual excitement. There was no change in the character of these attacks, nor in his expression, memory,

or behavior. He never manifested definite paralysis of his legs, though posterior weakness was quite manifest immediately before and after all of these convulsive attacks. Examination of his eyes failed to reveal definite lesions in the fundi until shortly before his death, at which time edema of the optic papilli was noted. His disease was diagnosed internal hydrocephalus.

This dog died suddenly from a perforation of the esophagus caused by a sharp bone. He was brought to me for examination. His thoracic and abdominal organs revealed no lesions which might account for his convulsive seizure. However, examination of his brain and spinal cord showed marked dilatation of the left lateral ventricle (fig. 5). The cause of obstruction was an ancient inflammatory adhesive lesion in the floor of the left lateral ventricle.

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Why Milk Is Safe for Babies

Efficiency comes to the barnyard. These five words summarize an article published in the July issue of *The Reader's Digest* under the title, "Why Milk Is Safe for Babies," by Dr. Guy G. Stevens (Corn. '11), of Groton, N. Y. The article was a condensed version of a story published in the June issue of *The Forum*, and relates the story of the long struggle in New York State to clean up the milk supply. The rôle played by the veterinarian in removing diseased cows from milking herds comes in for a good play. Dr. Stevens is to be congratulated for presenting these facts to the reading public, first in *The Forum*, and later in *The Reader's Digest*.

73rd Annual Convention A. V. M. A.
Columbus, Ohio, August 11-12-13-14

THE PATHOLOGY OF CROTALARIA SPECTABILIS ROTH POISONING IN CATTLE*

By D. A. SANDERS, A. L. SHEALY *and* M. W. EMMEL
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During January, 1934, attention was called to a chronic condition occurring in a herd of 65 adult beef cattle in which the outstanding clinical symptoms consisted of impaired appetite, ascites, diarrhea, weakness and tenesmus, with partial eversion of the rectum. Out of twelve clinical cases which developed over a period of six months, only one survived. The autopsy findings characteristic of these cases consisted of hydroperitonia, a firm indurated liver, enlarged gall-bladder, slight icterus, and edema of the abomasal folds, the loose tissues surrounding the duodenum and the double elliptical coils of the colon. A definite diagnosis could not be made from the symptoms and lesions shown by the affected animals. The history of these animals showed that they had been grazing on *Crotalaria spectabilis* Roth several months previously. Since some of the lesions observed were quite similar to those found in other animals in *C. spectabilis* poisoning by workers at this station, this condition was immediately suspected. Emmel, Sanders and Henley[1] found that the characteristic lesions of *C. spectabilis* poisoning in swine were indurated liver, ascites and edema.

Among many species of leguminous plants of the genus *Crotalaria* utilized as cover crops, *C. spectabilis* has a wide distribution in the southern part of the United States. Several species of *Crotalaria* have been incriminated as being poisonous to cattle. Stalker[2] found *C. sagittalis* L. poisonous for cattle and horses, and *C. burkeana* Benth was found by Theiler[3] to produce laminitis in cattle. Thomas,[4] working with poultry, was first to recognize the poisonous action of *C. spectabilis*. Thomas, Neal and Ahmann[5] observed that *C. spectabilis* was toxic to poultry and cattle. Neal, Ahmann and Rusoff[6] isolated an alkaloid from this plant and later Becker, Neal, Arnold and Shealy[7] reported that *C. spectabilis* was definitely toxic to cattle. Since the observations of these investigators on *C. spectabilis* poisoning in cattle dealt only with the acute cases, it was deemed important to study the chronic type of this condition. Experiments were therefore conducted to deter-

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mine the symptoms and lesions of *C. spectabilis* poisoning in cattle.

Experimental Observations

A steer twelve months of age and two cows four years of age which had not been exposed to crotalaria were given 3, 12 and 18 pounds, respectively, of finely ground mature *C. spectabilis* seed in a drench. The initial amount of material administered to the steer was 20 grams and to the cows 50 grams. The amounts administered were gradually increased until the entire quantities were consumed. The period of administration for the steer was 30 days. At the end of this period, the feeding of crotalaria was discontinued and the animal was held under observation for an additional 30-day period. The materials was administered to the cows over a 60-day period, and at the end of that time a post-mortem examination was made. During the experiment, the animals were given a feed consisting of ground corn, oats, bran, and timothy hay. In addition, the animals were allowed to graze, thereby paralleling in a large measure the natural conditions existing in the herd where the losses occurred. A control animal handled similarly, but receiving no *C. spectabilis*, remained normal.

Clinical symptoms: The clinical symptoms varied somewhat, according to the amount of material administered. The steer which received a comparatively small quantity of *C. spectabilis* seed exhibited a slight icteric discoloration of the sclerotica. This animal also developed tenesmus with partial eversion of the rectum and diarrhea. These symptoms were very characteristic of the field cases. The cows which received the larger dosages developed symptoms of salivation, became depressed and exhibited an unsteady gait. The scleral and palpebral portions of the conjunctivae, the unpigmented portion of the skin, and the feces showed yellowish discoloration. Purpuric areas 2.5 cm in diameter appeared in the outer integument over the lateral and ventral cervical regions. The skin of these regions was crepitant, indicating subcutaneous emphysema. The symptoms gradually increased in severity after approximately a ten-day period and became grave at the end of the observation periods. During the latter part of the experiment, all three animals became emaciated. The animals were destroyed for gross and microscopic study.

Gross lesions: In all of the experiment animals, the subcutaneous connective tissues of the entire body were of a distinct lemon or orange color and showed congestion with edema in the ventral cervical region. The walls of the abomasum were edematous. Extensive subserous petechiae and ecchymoses and hemor-

rhage into the lumen of the small intestine occurred. The omentum and mesentery were icteric and contained numerous petechiae. The mesenteric lymph-nodes were swollen and icteric.

The liver was firm, and of an intense greenish color. Upon cross section, the deep greenish discoloration was diffuse throughout the organ. The gall-bladder was greatly distended and large hemorrhagic erosions occurred on the mucous membrane. The bile was dark and thick. The kidneys were congested, the renal capsule tense and petechiae occurred in the pyramids. The mucous membrane of the urinary bladder showed petechiae. The spleen was enlarged, the capsule tense and on section appeared dark red in color. Petechiae occurred in the pancreas.

The thoracic organs were icteric. The lungs appeared variegated, with areas of congestion and edema interspersed with areas of interstitial and vesicular emphysema. Collections of air could be seen beneath the unruptured pleura. Much of the escaped air moved toward the hilus of the lung, gained the mediastinum, passed into the subcutaneous tissues at the entrance of the thorax from where it spread over the lateral cervical region. The mediastinal lymphnodes were edematous. A hypertrophy of the ventricles of the heart occurred in one case, while the other presented subendocardial ecchymoses with petechiae at the base of the pericardium.

Microscopic lesions: The lymphatics in the intestinal tract were found to be distended. The epithelial cells of the mucous membrane of the intestine showed an advanced stage of hydropic degeneration. The meshes of the reticular framework of the lamina propria were widely dilated. The lymphatics of the muscularis mucosa and submucosa were distinctly prominent. Lymphatic and venous distention was pronounced in the submucosa and muscularis mucosa of the wall of the abomasum. The interglandular capillary system of the mucous membrane of the abomasum was congested. The mesenteric lymph-nodes showed marked distention of the veins and capillaries, edema, slight fibrous tissue formation and pigmentation.

The liver substance in the experiment animals showed a varying degree of passive congestion, edema and connective tissue proliferation. The portal vessels were congested with resultant perivascular edema. In some instances the central vein of the hepatic lobule was completely filled with blood-cells, and there occurred marked distention of the capillaries. The radial cell cords were greatly compressed and atrophied and in many instances reduced to mere strands. This resulted in the replace-

ment by blood-cells of the spaces normally occupied by hepatic cells.

Interlobular connective tissue proliferation with round cell infiltration was in evidence around the bile-ducts and portal veins. In many instances the connective tissue replacement was seen to encroach upon the periphery of the hepatic lobule. Considerable pigmentation occurred throughout the organ. Marked pyknosis, atrophic degeneration, fragmentation of the hepatic cells, edema and hemorrhage occurred throughout the liver substance. The portal vessels in some instances showed marked congestion. Large confluent hemorrhages occurred in the mucous membrane of the wall of the gall-bladder; the blood-vessels, capillaries and lymphatics of the mucous and interstitial tissue were prominently distended and edema occurred throughout the intervening tissues; pyknosis and cellular degeneration and fragmentation were common.

The capillaries in the glomerular tufts were filled with erythrocytes. The epithelial cells were swollen, edematous and, in some instances, slightly exfoliated. The epithelium of the renal tubules showed an advanced stage of granular degeneration and fragmentation. In the midzonal region, venule and capillary congestion, perivascular edema and exfoliation of the tubular epithelium were prominent.

The splenic capsule was thickened and contained foci of capillary congestion. The splenic pulp was congested and perivasular edema of the malpighian corpuscles occurred. Slight proliferation of connective tissue appeared throughout the organ.

Congestion and perivascular and interstitial edema with foci of necrosis were present in the pancreas. The blood-vessels of the subepithelial lining of the pancreatic duct were congested and edema of the surrounding loose tissue was pronounced.

Rupture of the alveolar walls of the air vesicles of the lungs permitted the escape of air into the interstitial tissues. In the lobules thus affected, a varying degree of atrophy appeared. In the dark congested areas that constituted a significant portion of the lung tissue, the capillaries of the alveolar walls were engorged, thickened and tortuous. Many alveoli, being in addition already much compressed by large adjacent collections of interstitial emphysema, were occluded. Rupture of the alveolar wall, with subsequent hemorrhage, resulted in many instances.

Diffuse subendocardial hemorrhages, with interstitial congestion of the myocardium and hypertrophy occurred in the left ventricle.

Field Observations

The lesions occurring in field cases of chronic *C. spectabilis* poisoning were of longer standing than those observed in the animals fed experimentally. The liver was in an advanced stage of cirrhosis, presented a gray appearance and was very resistant to section. Edematous infiltrations were more pronounced. The serous covering of the enlarged gall-bladder was transparent, due to subserous accumulations of a clear, yellowish, fluctuating transudate. The abomasal cavity was largely obliterated by edematous infiltration of the spiral folds. The serous and subserous tissues surrounding the duodenum and double elliptical coils of the colon appeared as a greatly enlarged transparent membrane infiltrated with a clear, yellowish, fluctuating fluid. In many cases hydroperitonia was a prominent lesion.

Microscopically the lymphatics throughout the intestinal tract of the naturally occurring chronic cases showed a more pronounced distention. Edematous transudate accumulated and formed large expansions in the intercellular spaces of the epithelial cells in the spiral folds of the abomasum. The epithelial cells in these instances were greatly disfigured, pushed aside and partly exfoliated. Large bands of fibrous tissue replacement occurred throughout the splenic substance. The liver showed extensive thickening of the capsule and reticular framework. The radial cell cords were widely separated and compressed by fibrous tissue. The alveolar walls in many of the lobules of the lungs were thickened and indurated by connective tissue proliferation.

Development of field cases: The above observations lead to the belief that *C. spectabilis* was the causative agent responsible for the losses incurred in the herd previously mentioned. It has been observed that *C. spectabilis* is usually not palatable to live stock except when in blossom and ordinarily will not be eaten under field conditions unless other feed is scarce. These animals gained access to a five-acre field of mature *C. spectabilis* in November, 1933. It should be stated that the pasture on which this herd was accustomed to graze was flooded and that the animals were removed to higher soil on which *C. spectabilis* constituted a significant portion of the plant life. The herd consumed the available forage on this area during a period of two weeks of grazing, after which the animals were removed to pastures free from crotalaria.

During the second month following the exposure, three animals died after being noticeably ill for two weeks; four animals died during the third month after ailing noticeably for a similar

period. During the fourth, fifth and sixth months following exposure, five cases developed with four fatalities. One adult cow manifested clinical symptoms of crotalaria poisoning for several weeks, but survived in a very emaciated condition. With these long incipient periods and latency of clinical manifestations, the etiological agent may easily be overlooked and some secondary factor involved in the alterations may be suspected as the cause of the condition. It is necessary, therefore, to obtain a careful history of the animals in order to determine the underlying cause of the trouble.

SUMMARY

1. Clinical symptoms, gross and microscopic lesions occurring in a herd of cattle under natural conditions were reproduced in animals experimentally by daily oral administration of pulverized *Crotalaria spectabilis* seed.

2. *C. spectabilis* poisoning, as observed in cattle under natural conditions, assumed a chronic form with gradual onset unaccompanied by clinical manifestations in the beginning.

3. Clinical symptoms in cattle grazing daily on crotalaria for two weeks developed after a latent period of from two to six months. These consisted of impaired appetite, diarrhea, ascites, acute bloating, decubitus and tenesmus, with partial eversion of the rectum.

4. At autopsy there were observed: hydroperitonia; a firm, resistant, indurated liver; enlarged gall-bladder; icterus; congestion of the kidneys; congestion, edema and emphysema of the lungs; edematous infiltration of the submucous layer of the abomasal folds and of the subserous coverings of the double elliptical coils of the colon; with hypertrophy of the cardiac muscle.

5. Microscopically there was observed a diffuse inter- and intralobular fibrosis, atrophic degeneration, and pigmentation of the hepatic parenchyma; fibrosis of the spleen; parenchymatous degeneration and fragmentation of the renal epithelium; connective tissue proliferation, edema and emphysema of the pulmonary tissues; and parenchymatous degeneration and epithelial exfoliation of the mucous membrane of the intestinal tract.

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Watch Out for This Party

A party who gives his name as O. W. Danziger and his address as Kansas City, Mo., has been reported from several western states, from California to Nebraska, as calling on veterinarians and taking orders for army veterinary outfits. In most instances, this party collects a down payment with the understanding that the balance is to be paid at a later date. This is the last that the veterinarian ever sees of Danziger and, of course, the goods fail to arrive. Veterinarians are cautioned to be on the watch for this party. A word to the wise is sufficient.

McKINLEY MONUMENT AND HUNTINGTON BANK BUILDING, COLUMBUS

OAK POISONING IN RANGE CATTLE AND SHEEP*

By I. B. BOUGHTON *and* W. T. HARDY
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During the month of April, 1935, the writers observed several outbreaks of a highly fatal poisoning among range sheep and cattle resulting from the eating of the buds, green shoots and young leaves of the common shin oak (*Quercus breviloba*), which grows abundantly on the ranges of the Edwards Plateau region of West Texas. Personal investigations and reliable reports from ranchmen in the county where this Station is located showed that 502 cattle and 426 sheep died from this condition on 38 different ranches within a period of about four weeks. The sheep losses were undoubtedly greater than the number mentioned, inasmuch as we were unable to get complete data regarding the mortality in this species. Reports from other sections, where this and related species of shin oak are abundant on the range, indicated that the losses there were correspondingly heavy.

As far as we were able to determine, from personal investigation and reliable reports, the mortality of affected animals averaged about 85 per cent in cattle and 90 to 95 per cent in sheep. One man, who lost in the neighborhood of 100 sheep, stated that all his clinically affected animals died.

LITERATURE

Mascal,[1] in 1662, mentioned the toxicity of oak leaves for cattle and Cornevin,[2] in 1893, reported the results of a rather extensive study of the subject, describing the symptoms and discussing the possible rôle of tannic acid (or soluble tannates) in causing the condition. Glover and Robbins[3] mention the toxicity of oak leaves in their bulletin. Marsh, Clawson and Marsh[4] reproduced the poisoning experimentally in cattle by feeding the buds and young leaves of shin oak (*Q. havardi*), which is common in the extreme western parts of Texas. No definite reports of the occurrence of the poisoning among range sheep seem to have been made as far as a search of the available literature shows.

The hemoglobinuria mentioned by Cornevin was not observed by Marsh, Clawson and Marsh, nor did the present writers see any evidence of it in the range outbreaks reported herein.

*Received for publication, January 9, 1936.

CAUSE

The cause of the poisoning is not definitely known. Cornevin indicates that tannic acid (or tannates) in the buds, shoots and young green leaves is the etiologic agent but Marsh, Clawson and Marsh were unable to reproduce the condition in two cattle which received large daily doses of this drug for several weeks. The writers were unable to poison a healthy mutton sheep which received daily doses of 30 grams of tannic acid for 37 consecutive days.

The fact that the poisoning occurs almost every year in a few cattle in this section, when the shin oak buds and flowers, indicates that the toxic principal is always present, either in the buds, shoots or young green leaves (or in all of them) at this time of the year. But the fact that severe outbreaks occur in this region only following winters of severe drouth and consequent short, dry range, indicates that there is considerable variation in the toxicity from year to year. It should also be borne in mind that range live stock are starved for green feed during such years and probably consume exceptionally large quantities of shin oak, often to the exclusion of other available feeds.

Cattle and sheep of all ages were affected. Our observations indicate that the animals apparently eat a considerable quantity of the material over a period of several days before clinical symptoms are manifested. On one ranch, where the cattle had been on feed in a small enclosure for more than a month, the first affected cow was noticed exactly eleven days after the animals had been turned into a pasture where the shin oak was abundant. We were unable to get definite information as to the time period in any of the sheep outbreaks, so must assume that it corresponds, relatively, to that observed in cattle. As a rule, few cases develop later than eight days after the animals are placed on feed away from the brush but we did observe one typical case which developed 14 days after the animal was penned.

Animals which have been forced to rustle for their food on the range all winter do not seem to be affected so often as do those animals which have been on feed for several weeks previous to the time they are turned on the range and feeding stopped. Evidently such animals are ravenous for green feed and eat large quantities of the succulent buds, shoots and leaves.

Conversation with many of the older ranchmen, who have been running sheep in this region for 30 to 40 years, disclosed the fact that most of them were familiar with the condition in cattle but had never seen it in range sheep before the present year.

In all probability the condition has not appeared in these animals during previous budding seasons because there has always been some available ground vegetation. During the present spring, however, grass and weeds have been conspicuous by their absence on many ranches. Consequently the sheep were forced to browse on the brush. The shin oak often grows to a height of 15 feet or more but there is invariably a thicket of second-growth brush, easily reached by sheep, around the base of such trees.

Just how long the buds, shoots and young leaves remain toxic for live stock is not known, but practical experience has shown that animals can safely be turned into shin oak pastures when the leaves have deepened in color, losing their tender green tint. Naturally this period varies with the season; in some years practically all the shin oak trees bud and flower about the same time. During the present season, however, the budding process continued more than a month as a result of variation in the moisture conditions. We have observed in the same pasture trees in full leaf, trees on which the buds are just appearing, and trees which showed no signs of budding. Obviously the danger period in such a pasture will be prolonged.

Course

At the beginning of the outbreak, the disease seemed to be most severe and ran a fairly acute course. A few animals died within 24 hours after manifesting symptoms, but the majority manifested illness five to ten days before succumbing. Some chronically affected cattle lingered for more than a month. Most of the affected sheep suffered from the more acute type of poisoning and succumbed within one to three days after manifesting symptoms. Complete recovery, of either cattle or sheep, seldom occurred under two or three weeks.

Symptoms

Typically affected cattle show listlessness, complete inappetance accompanied by cessation of rumination, a brownish, sticky nasal discharge, sunken eyes, a rough hair-coat, "tucked up" belly and an obstinate constipation. The continued efforts at defecation, resulting in the passing of small, hard, brownish-black balls of mucus- or blood-covered feces, are almost constantly observed. The pulse is fast and wiry, the respirations are markedly increased in rate and shallow in character, while the temperature is either normal or slightly below. Cold, edematous swellings in the subcutis of the pendant portions of the body were seen frequently.

Close observation of typically affected cattle showed that they drank small quantities of water at very frequent intervals. The frequent passage of abnormal quantities of a clear urine of low specific gravity was characteristic. We did not see any affected animals which showed either hematuria or hemoglobinuria.

In very acute cases, which resulted fatally in 24 to 36 hours, the symptoms were increased in severity. In most fatal cases the obstinate constipation is followed, in from one to ten days or so, by a fetid diarrhea. At first the forcibly-ejected feces show much mucus and later are composed of almost pure blood.

In our observations of more than 100 cases in cattle, a few animals, from the onset of the sickness, showed bloating and a fetid diarrhea with the feces containing shreds of mucus and blood. These cases were invariably fatal.

The symptoms observed in affected sheep showed no significant difference from those manifested by cattle. The edematous swelling along the belly was an almost constant symptom. It was noted that sheep seemed to be more severely affected on bright, hot days.

Postmortem Lesions

The findings at autopsies of typical cases were markedly constant. The edematous swellings were full of a clear, jelly-like material with no evidence of hemorrhages. Masses of this material were encountered in the abdominal and pelvic cavities, as well as infiltrating the mesentery and the omentum. Large quantities of clear fluid were almost constantly present in both the belly cavity and the pericardial sac. Many of the lymph-glands were edematous and flabby. The normal-appearing bladder was distended with a very clear urine; the kidneys pale and studded with pinpoint hemorrhages; the liver friable, slightly enlarged and of a purplish color; the gall-bladder invariably distended with a thick, brownish bile containing small, putty-like brown flakes. No gross lesions were observed in the spleen.

The first three stomachs showed no gross pathological changes but the omasum was usually completely stuffed with exceedingly dry ingesta. The abomasum was either pale and edematous or else showed a severe hemorrhagic gastritis. The same conditions were noted throughout the small intestine. The edematous condition of the mucosa was very common in sheep while the severe gastritis and enteritis was seen in almost every autopsy in cattle. The lining of the cecum and the large intestine was often covered with a thick, black, false membrane which, when re-

moved, exposed the red, eroded and hemorrhagic mucous membrane underneath. The feces in the colon were composed of either small, hard, black, mucus-covered balls or else a semi-liquid, fetid, molasses-colored mass containing strings of mucus and a considerable admixture of blood.

The heart musculature was usually pale and flabby, while the fat around the base showed a pinkish tinge. In many cases no gross lesions were found in the lungs. Occasionally pinpoint hemorrhages were seen sprinkled over the surface of these organs. Characteristically, in sheep, the venous blood was cherry red in color and coagulated much more quickly than does normal blood.

Treatment and Prevention

No efficient means of treatment has been found. The administration of physics, saline or oleaginous, in appropriate doses accompanied by high enemas of warm, soapy water, may occasionally be of some value in mild cases but nothing in the way of medicinal treatment has been found which benefits typical severe cases.

The alkaloidal physics, pilocarpine and arecoline, in either small, repeated doses or large single doses, are likewise of no value in relieving the constipation. Observation in these outbreaks showed that by far the great majority of animals which had a complete loss of appetite succumbed to the disease.

The reports of all workers with this disease show similar unsatisfactory results from medicinal treatment. Marsh, Clawson and Marsh[3] report that the daily experimental administration of Epsom salts to two cattle kept the feces soft but did not prevent the appearance of typical poisoning. These authors conclude that the constipation is not the principal factor in this disease. Our experience in the outbreaks here discussed confirms this belief.

In animals which still have some appetite the liberal feeding of bright alfalfa hay is usually attended by complete recovery within a reasonable period of time. Obviously the animals should be penned and put on feed, especially hay, as soon as any cases are seen.

Marsh, Clawson and Marsh demonstrated that mature leaves are toxic but to a lesser degree than are the young leaves and buds. Poisoning from the ingestion of mature leaves has never been observed in this region. Evidently there is enough other range vegetation available at this time to preclude an exclusive diet of mature shin oak leaves.

Prevention of the condition, in this section, should provide for the separation of the animal and the shin oak during the period when the buds and young leaves are apparently very toxic.

On some ranches the animals can easily be herded away from the brush but such a practice is not practical on many ranches where the brush is abundant. Practically, on the range in some parts of West Texas, where the small shin oak (*Q. havardi*) is abundant, it has been shown that animals can be allowed to eat the brush provided they are receiving a liberal amount of good hay (preferably alfalfa) every day. Experience in this region where the outbreaks discussed occurred, and which is thickly dotted with various kinds of fairly tall brush in addition to shin oak, however, has shown that prevention is more easily and economically brought about by pen feeding on hay and some concentrate while the shin oak is budding and flowering.

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The Cow Tester's Dream

You ought to be with me,
And see the fun,
And how I keep
The cows on the run.

They buck and kick
And bawl and stall;
Put the ropes on them
And see them fall.

Put them in stanchions,
And have them secure.
Then the operation,
They must endure.

Turn them loose
To the fields to roam,
Now my work's done,
I am going home.

F. J. MUECKE

To Stop Rooting

To stop pigs from rooting up pasture, feed them an animal protein, tankage or skim milk with calcium and phosphorus. Such a ration, with rings in the noses of the pigs, will reduce the rooting to a minimum.

—*American Hampshire Herdsman.*

EXPERIMENTAL VACCINATION OF RANGE CALVES WITH A LIVING CULTURE OF BRUCELLA ABORTUS

I. Results of Agglutination Tests Made at Intervals During Three Years Following Vaccination*

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and HADLEIGH MARSH, *Bozeman, Mont.*
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In 1932, arrangements were made with the United States Bureau of Animal Industry to carry out an experiment to test the efficacy of calfhood vaccination against Bang's disease in a herd of range cattle, using the living culture of a strain of *Brucella abortus* of low virulence prepared in the laboratory of the Experiment Station of the U. S. Bureau of Animal Industry.

The herd selected for the experiment consisted of about 1,500 grade Hereford range cattle located in Powder River County. These cattle are run on grass most of the time, both summer and winter, but the range is supplemented with hay feeding to some extent in the winter, when shortage of grass or severe weather conditions make hay feeding necessary. During the winter of 1931-1932, there was a heavy loss from abortion, the diagnosis of Bang's disease having been established by the agglutination test.

The heifer calves held for replacements in 1932 and 1933 were used for the experiment. The total number of heifers in the experiment was 415. The first lot of 109 heifers vaccinated were older than is recommended for calfhood vaccination, ranging from six months to more than one year in age. The remaining 306 animals were between three and six months old. Of the total 415 heifers, 295 were vaccinated and 120 were left unvaccinated as controls. All the calves were tattooed with serial numbers, and they were also branded with marks which distinguished the vaccinated from the control animals.

Blood samples were taken from all the calves before vaccination. Of the 109 older heifers, 17 showed some degree of agglutination at 1:25, six reacted completely at 1:50, and one showed complete agglutination at 1:100. Of the 306 calves under six months old, six reacted to some extent, only one agglutinating completely at 1:100.

*Contribution from the Montana Veterinary Research Laboratory and the Montana Livestock Sanitary Board. Paper No. 69, Journal Series, Montana Experiment Station. Received for publication, February 11, 1936.

The vaccination consisted of the subcutaneous injection of 10 cc of the live culture vaccine of low virulence supplied to us by the U. S. Bureau of Animal Industry.

This report is concerned only with the results of agglutination tests on the blood serum of the heifers at intervals during three years following vaccination. The question of the development of immunity as the result of vaccination will be the subject of a later report.

The agglutination tests following vaccination have been made at somewhat irregular intervals on the first two lots of calves placed in the experiment, consisting of 363 animals. It has not been possible to test all the cattle on every test. This has been partly due to the fact that in the range operation a complete gather was not always possible. A major interference with the progress of the experiment occurred in 1934, when, due to the severe drouth, the owner of the cattle was obliged to cut his herd from 1,500 to 400 cattle. He retained only about 190 of the heifers on experiment. The result is that we have a record of two tests following vaccination on the majority of the heifers, covering a period of one year, and on the 190 retained we have a record of five tests, covering a period of three years.

For the purposes of this report, the reactions of only the vaccinated heifers are discussed, leaving the controls out of consideration. It is necessary to consider these reactions in two groups, due to the difference in age at the time of vaccination, and also due to the fact that the first post-vaccination test was made three months after vaccination in the first group, and eight months after vaccination in the second group. Of the 109 heifers in group 1, 76 were vaccinated. On the first post-vaccination test, 75 of the vaccinated heifers were tested, while on subsequent tests the number available varied from 43 to 47. Of the 254 heifers in group 2, 177 were vaccinated, of which the number available for post-vaccination tests varied from 154 to 88.

Tables I and II show the agglutination titres of the vaccinated heifers in groups 1 and 2, respectively, at five intervals after vaccination. The total number of cattle tested at each time is shown, with the number and percentage in five classifications as to titre. In the negative column are shown all cattle that were completely negative at 1:25 or showed only partial agglutination at that dilution. In the four other columns are shown the numbers and percentages showing titres of 1:25, 1:50, 1:100 and 1:200, as indicated by the maximum dilution at which complete agglutination occurred.

The figures tabulated in tables I and II are shown graphically

TABLE I—*Agglutination titres of heifers in group 1, vaccinated at age of 6 to 12 months.*

Interval Between Vaccination and Test (months)	Heifers Tested	Negative		Titre 1:25		Titre 1:50		Titre 1:100		Titre 1:200	
		No.	%	No.	%	No.	%	No.	%	No.	%
3	75	10	13	17	22	24	31	0	0	24	33
12	46	9	20	16	35	16	35	5	11	0	0
28	47	36	77	11	19	2	5	0	0	0	0
31	43	30	70	8	19	5	11	0	0	0	0
38	47	29	62	15	32	3	6	0	0	0	0

TABLE II — *Agglutination titres of heifers in group 2, vaccinated at age of 4 to 6 months.*

Interval Between Vaccination and Test (months)	Heifers Tested	Negative		Titre 1:25		Titre 1:50		Titre 1:100		Titre 1:200	
		No.	%	No.	%	No.	%	No.	%	No.	%
8	141	67	48	43	30	23	16	8	6	0	0
13	154	74	48	64	42	13	8	3	2	0	0
25	91	88	97	2	2	1	1	0	0	0	0
28	88	82	93	5	6	0	0	1	1	0	0
35	92	86	93	5	6	1	1	0	0	0	0

in the chart (fig. 1). In making the graphs, percentage figures were used, as the number of animals tested varied. In the first series of graphs representing the classified reactions of group 1, it will be seen that at three months after vaccination only 13 per cent of the heifers were negative at 1:25, and 33 per cent reacted at 1:200 or higher. At twelve months, there were no reactions at 1:200, but 80 per cent of the heifers reacted at 1:25, 1:50 and 1:100. At 28 months, 77 per cent of these heifers were negative at 1:25, and none reacted as high as 1:100. There was little change on the two subsequent tests. At 38 months, the percentage of reactions at 1:25 increased to 32, but this change is considered to be insignificant, as it has been found, in repeatedly testing uninfected cows in infected herds, that agglutination at 1:25 may appear and disappear in the same animal in a series of tests, without any significance as to the development of actual infection.

The second series of graphs shows the classified reactions of group 2. In this lot the first post-vaccination test was made eight months after vaccination, and at that time there were no reactions as high as 1:200, while 48 per cent were negative at 1:25. At 13 months, the percentage of negatives was the same, but the percentage of reactors at 1:25 was increased at the expense of the 1:50 and 1:100 groups. At 25 months, 97 per cent of these heifers were negative, and there were no reactions as high as 1:100. There was practically no change in these percentages at 28 and 35 months. At 28 months, one animal showed a titre of 1:100, but this heifer dropped back to 1:50 on the last test.

Comparing the results of the tests for groups 1 and 2, there is evident in the graphs a difference between the two lots, which is probably correlated with the difference in the ages of the two groups at the time of vaccination. The heifers in group 1 were all over six months, while those in group 2 were between four and six months of age. A comparison of the graphs shows that the heifers in group 2 lost their reactions more rapidly than those in group 1, and that a larger percentage of low-titre reactions persisted in group 1 than in group 2.

A fact which should be considered in connection with this report is that all the heifers involved in this experiment went through at least one period of pregnancy during the period covered by this series of tests. As none of the later tests showed sufficiently high titres in any of the heifers to indicate active infection, it is evident that *Br. abortus* did not become established in any of the animals as a result of vaccination with a living cul-

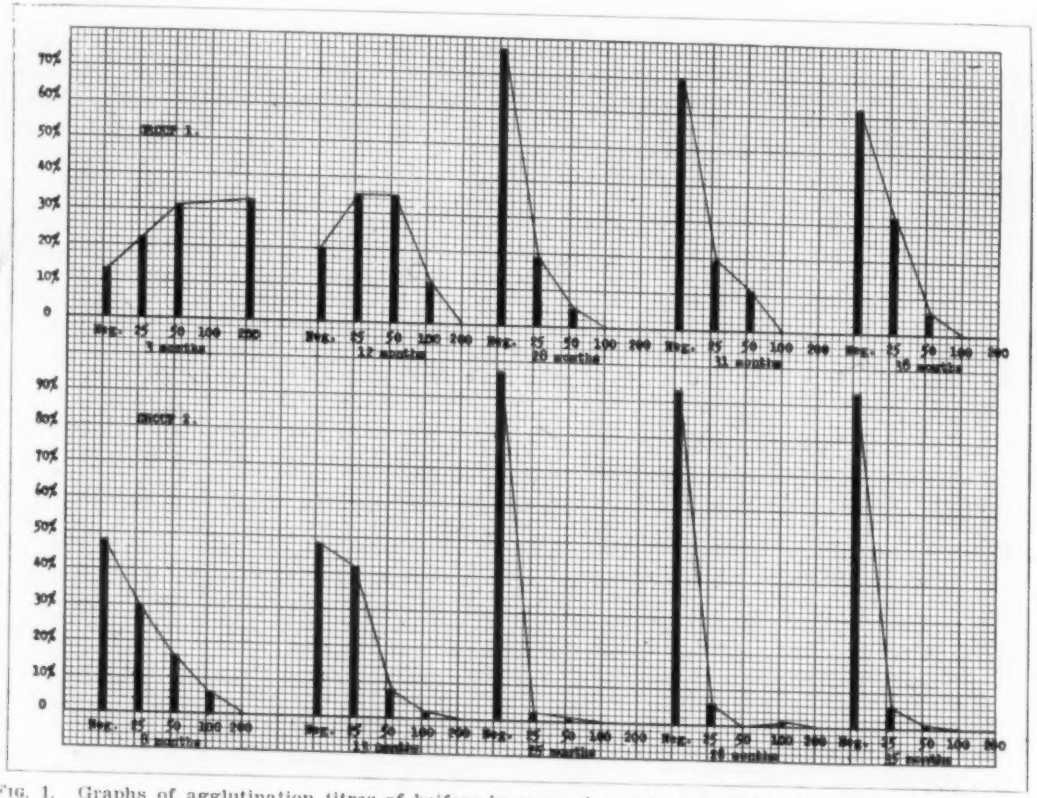

FIG. 1. Graphs of agglutination titres of heifers in group 1, vaccinated at 6 to 12 months, and group 2, vaccinated at 4 to 6 months.

ture of low virulence. This is true although a number of the heifers in group 1 were sexually mature at the time of vaccination.

SUMMARY

This report records the results of post-vaccination tests for *Br. abortus* agglutinins in the blood serum of range heifers vaccinated with a living culture of *Br. abortus* of low virulence. These tests show that in a group of heifers vaccinated at six to twelve months of age, the agglutinins produced by vaccination were reduced in twelve months to the extent that no reactions occurred at a 1:200 dilution, and that at the end of two years, 77 per cent of the heifers were negative at 1:25, and none reacted at a dilution higher than 1:50.

In a group of heifers in the same herd which were vaccinated at the age of four to six months, there were no reactions at 1:200 in eight months, and at the end of two years, 97 per cent of the heifers were negative at 1:25, with no reactions higher than 1:50.

The results of these agglutination tests also indicate that *Br. abortus* did not become established in any of the heifers as a result of vaccination.

Phi Zeta Initiation at Iowa State College

The annual initiation ceremony and banquet of Gamma Chapter of Phi Zeta, national veterinary honor society, was held at the Memorial Union of Iowa State College, May 12, 1936. Twelve active members were received into the Society and were guests of honor at the banquet that followed the initiation.

Eight members of the senior class of the Division of Veterinary Medicine were elected to active membership: Clyde F. Cairy, Wm. T. Dunn, Albert C. Emminger, Glenn C. Holm, Carl E. Venzke, Herbert P. Wessels, Frank B. Wilkinson and Stanley N. Wood. The three members of the junior class elected to active membership were: Howard C. Raven, Benjamin Rosenfeld and Piao Sheo. Dr. Ernest F. Waller, instructor in the Veterinary Pathology Department, Iowa State College, also was elected to active membership.

Dr. Chas. Murray, president of Gamma Chapter, presided as toastmaster at the banquet. Dr. H. D. Bergman, of the veterinary faculty, gave the formal address, "Progress in Modern Medicine," stressing particularly recent achievements in medicine that indicate real medical progress as distinguished from passing fancies in the form of medical fashions and fads.

AN ATTEMPT TO TRANSMIT ANAPLASMOSIS BY BITING FLIES*

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INTRODUCTION

Due to the importance of anaplasmosis in the South, particularly in Louisiana, it seems that these results should be reported in brief form, even though the work was done some time ago. During the past two years, this disease apparently has been on the increase in Louisiana. In areas that have been released from the Texas fever scourge recently, it may be that the disease is no more prevalent than before, but it is now being recognized when previously it was mistaken for tick fever.

Late in 1929, 15 large, black, blood-sucking flies (*Tabanus atratus*) were allowed to feed intermittently on an active case of anaplasmosis and then on a susceptible Jersey cow. On the 25th day after the first feeding, marginal dots appeared in the erythrocytes and a temperature rise of 2° was recorded, although the cow did not present a good clinical case of the disease. Marginal dots appeared at intervals of about 16 days for a period of several months.

The afore-mentioned observations, with reports in the literature, led to a set-up of controlled experiments which were carried out during the three succeeding summers.

REVIEW OF LITERATURE

Most of the literature on the subject reported was published during the course of this experiment, while some of it has been published since the work at the Louisiana Station was completed. Nevertheless, the work of Dr. Morris seems worthy of report.

In the 1926-30 report of the Oklahoma Station, Sanborn[1] stated that in Oklahoma the castor-bean tick (*Ixodes ricinus* Linn.) was found to transmit the disease. He referred to the dehorning saw also as a carrier. He suggested that such flies as the brown horse fly, *Tabanus sulcifrons* Macquart; the stable fly, *Stomoxys calcitrans* Linnaeus; and the horn fly, *Haematobia irritans* Linnaeus, were prevalent and should be investigated.

Sanborn, Stiles, Moe and Orr[2] reported in the same publication the results of allowing twelve *Tabanus sulcifrons* to feed on an

*Received for publication, March 23, 1936.
†Deceased May 4, 1934.

infected cow and then on a susceptible one. The latter did not come down in nine months, at which time she was given 16 cc of infective blood and developed a fatal case. Parasitized blood cells were found in the flies for 16 hours after they had fed on the carrier animal. A sick cow and two susceptible ones were taken 25 miles by truck in order to have easy access to flies. Flies were caught when feeding on the sick cow and transferred to a susceptible cow. One susceptible animal was exposed to the bites of three species of tabanids (*T. gracilis, T. sulcifrons, T. atratus*) and no symptoms developed in 120 days of observation. The other cow was exposed to 29 engorgements of *T. gracilis*, seven of *T. sulcifrons*, and five of *Chrysops sequax*. Suspicious dots appeared in 107 days and death occurred in 119 days, with typical symptoms of anaplasmosis.

Stiles,[3] in a general survey of the disease as to occurrence, symptoms, mortality, and lesions, also stated that many veterinarians in the areas involved were of the opinion that flies, mosquitoes and ticks were possible vectors.

Reese[4] showed that the tick, *Rhipicephalus sanguineus*, transmitted the disease during the nymphal stage. He[5] also transmitted the infection by means of a lancet used on infected and then on susceptible animals. This adds more to the belief that infected instruments may be a source of infection.

Stiles[6] reported that eight ticks had been incriminated, but mentions that foreign literature at that time had reported negative results with flies. Boynton[7] states that animals with acute cases or carriers may be kept in direct contact with susceptible animals with no danger from the cohabitation, and that transmission is apparently dependent upon mechanical means or intermediate hosts. Flies and ticks are mentioned as possible vectors.

Sanborn, Stiles and Moe[8] presented four positive transmissions. One susceptible cow was subjected to a total of 43 bites in 14 days. The flies used were *T. gracilis* Wied., *T. fuscicostatus* Hine, *T. sulcifrons* Macq., *T. venustus* O. S., and *Silvius pollinosa* Will. The first evidence of disease was noticed 39 days after the first feeding and death occurred on the 43rd day. A second cow was exposed to 79 bites of *T. gracilis* during a period of eight days. Anaplasmosis developed in a month, but the animal recovered. A third animal was subjected to 24 bites of *T. sulcifrons* and showed a mild clinical case in 66 days after the first feeding. The fourth cow had 115 bites from *T. venustus* over a period of 30 days. In 73 days, anaplasma dots appeared and in 81 days the temperature was very high. Recovery followed after about 14 days of acute

symptoms. *T. fuscicostatus* and *S. pollinosa* were not checked separately.

Sanders[9] demonstrated two positive transmissions. Two susceptible animals were put in closed quarters for the average period of incubation. One cow was exposed to the bites of 100 flies (*T. fumipennis* Wied.) after they had partially engorged on an acute case. Sixty-two days after the first feed, marginal dots appeared. This was a laboratory diagnosis and not a clinical case. Thirty days later, 15 cc of virulent blood did not produce the disease. Another cow was exposed for ten days to several hundred flies of species *Stomoxys calcitrans*. On the 42nd day from the start, dots appeared but it was not a clinical case. This animal likewise did not respond later to 15 cc of virulent blood. The negative response to virulent blood is ample proof of having suffered at least a mild case of anaplasmosis.

Reese[10] reported that eleven species of ticks, three of the genus *Boöphilus*, two of genus *Dermacentor*, one of genus *Hyalomma*, two of genus *Ixodes*, and three of genus *Rhipicephalus*, all of the family *Ixodidae*, had been definitely found capable of spreading the disease.

Du Toit[11] agrees closely with Reese on the tick situation, mentioning four of the genus *Boöphilus*, three of *Rhipicephalus*, one of *Hyalomma*, one *Ixodes*, and two of the genus *Dermacentor* as vectors. He cites instances of spread by instruments, but suggests that flies play no rôle as carriers in nature. He refers to the experimental transmission as reported by Sanborn and coworkers.[2]

Taylor[12] reports his work in England. He states that *T. gracilis*, *T. sulcifrons* and *T. venustus*, that were considered carriers by the Oklahoma workers, as well as *T. fuscicostatus*, *Chrysops sequax* and *Silvius pollinosa* observed in Oklahoma, are not present in England. He used flies of the genus *Haematopota*, closely related to *tabanids*.

A cow was given 20 cc of virulent blood and in 34 days she was sick. From the 34th to the 53rd day, horse flies (46 bites) of species *Haematopota pluvialis* fed on the sick animal and then on a susceptible one. At the close of 14 weeks, the latter had not shown symptoms of the disease.

Another cow was given 30 cc of blood and showed symptoms on the 26th day. From the 26th to the 43rd day, a susceptible cow was exposed to over 6,000 bites of the stable fly, *Stomoxys calcitrans*, that had first been on the sick cow. There was no response on the part of the susceptible cow.

Blood from the second acute case was also given to susceptible

animals in doses of 0.01, 0.1, 1, 10, and 20 cc. All animals but the one getting 0.01 cc contracted anaplasmosis after inoculation. The cow that did not respond to 0.01 cc and those not infected by the flies were later given 20 cc each of virulent blood and all developed the disease, proving they were susceptible. From these brief experiments, Taylor does not think the disease is spread by biting flies in that country.

Hine,[13] in 1907, had described many of the most common horse flies of Louisiana.

Method of Procedure

Two rooms in the Animal Pathology Building were tightly screened to prevent free passage of flies and mosquitoes. There is a solid wall between the two rooms, so it was necessary to go from one room to the other by a short hallway which also was screened. The sick or carrier animals were housed in one room and the flies were transported from the sick to the apparently susceptible host.

Flies were collected on a plantation about 15 miles southeast of Baton Rouge. In that section flies were very plentiful and could be easily caught.

The general plan of all experiments was to use six animals as follows:

1. A sick or recently recovered animal was used as the carrier.
2. A control animal was injected with blood of animal 1 to be sure that it was a carrier of this disease complex.
3. An animal upon which the recently caught flies were allowed to feed. This was an attempt to prove the flies were not carriers when brought in from the field.

4-6. Three animals exposed to the bites of flies after the flies had first been allowed access to the carrier.

At the close of the feeding period, the animals were observed for varying lengths of time. Blood-smears were stained and observed for the presence of anaplasma and at the same time the temperatures were recorded. At the termination of the period of observation, animals 3, 4, 5 and 6 were given inoculations of virulent blood to determine whether or not they were actually susceptible.

The animals used were purchased from the local market and no history of their previous exposure was available. It is very difficult to check the susceptibility of an animal because recovered animals sometimes do not show *Anaplasma marginale*. Thus the only positive proof of susceptibility is the response to an injection of positively infective blood.

Results

The first series of animals were exposed to the bite of *Tabanus atratus*, the large black blood-sucking fly, during the summer of 1930. Fifteen flies were allowed to start feeding on the carrier and they were then moved to the susceptible animals, each animal being exposed to the bites of 15 flies. During the 80-day period following, none of these animals (3, 4, 5 and 6) showed the presence of marginal dots or a rise in temperature.

When injected with virulent blood, all showed clinical cases of anaplasmosis in twelve to 15 days. These animals were inoculated with the virus from an animal carrying the Reese strain of anaplasmosis, which always shows a short incubation period in tests run at this station.

The next group, during the summer of 1931, was subjected to the bite of the horn fly *Haematobia irritans*. About 150 flies were permitted to feed on the infected animal, and were then transferred to a susceptible one. The flies were handled in screen cages. The feeding period extended over three days. A temperature chart was kept for 100 days and blood-smears were made frequently. At the close of the 100-day period no animal had shown evidence of anaplasmosis.

Animals 3, 4, 5 and 6 each received 50 cc of virulent blood. Three animals developed clinical cases of the disease, while one did not at the end of 50 days. This animal was to be observed up to 80 days, but the record of the outcome was lost and there is no one present in the department now who knows.

The third group, during the summer of 1932, was handled the same as the others, but the vector used was *Tabanus fuscicostatus* Hine. At least 75 flies were used on each susceptible animal and each fly was allowed to make three contacts. The flies were placed in small jars with screened tops. They were allowed to puncture the skin of the carrier and then were transferred to the susceptible animal. The fly-feeding process covered a period of a week.

This group of animals was observed for 80 days, temperatures and blood-smears being taken daily and blood counts weekly. At the end of the period, the animal exposed to the non-infected flies and the three others were still normal. These four animals, when inoculated with virulent blood, developed clinical cases of anaplasmosis.

Discussion

With the disease becoming more common in the state and live stock owners more conscious of its importance, it is apparent that something needs to be done along the line of treatment and pre-

vention. In connection with the prevention and control, it becomes necessary to know more about how the disease is spread.

Cases of anaplasmosis are very rare in this state from November 1 until about April 15 to May 1. This seems to be quite indicative that some carrier agent which is inactive during late fall, winter and early spring may be responsible for the spread. Generally cases are sporadic in nature, which indicates that the disease is one possessing a very low degree of contagiousness or ability to spread. On the other hand, it does at times occur in a large number of animals in a herd, acting like a highly contagious disease. We have no records on the occurrence of possible vectors present in these cases. These instances are reported by practitioners and their diagnoses are confirmed by the blood-picture, clinical picture, and postmortem pictures of those animals that die.

It will be observed that Dr. Morris had one case develop after *T. atratus* had gone from an infected to a carrier cow. In the controlled project, three cows exposed to the bite of this fly did not develop the disease. They were later proved to be susceptible. No explanation was attempted, except to state that possibly in the preliminary case some instrument such as bull tongs might have been used without having been sterilized before being transferred from the sick to the susceptible animal.

It will be noted that Dr. Morris did not have the same flies as reported carriers by Sanborn *et al.* or by Sanders. Sanborn and co-workers did use *T. fuscicostatus* with four other flies,[8] and *T. atratus* with two other flies,[2] obtaining positive results, but neither of them was used singly. This may be significant in accounting for the difference in results.

SUMMARY

1. Three animals exposed to intermittent feeding of the large, black, blood-sucking fly *(Tabanus atratus)* failed to develop the disease in 80 days. One preliminary test with this fly proved positive in 25 days.

2. Three animals exposed to intermittent feeding of the horn fly *(Haematobia irritans)* failed to develop the disease in 100 days.

3. Three animals exposed to intermittent feeding of *Tabanus fuscicostatus* Hine failed to develop symptoms in 80 days.

4. All nine exposed animals were known to be susceptible because all developed the disease when given intravenous injections of virulent blood.

5. No attempt is made to explain why these results are different from those reported from Oklahoma and Florida.

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OBSERVATIONS PERTAINING TO STANDARDS OF INTERPRETATION OF AGGLUTINATION TITRES IN THE DIAGNOSIS OF BANG'S DISEASE*†

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I. The Effect of Different Standards of Interpretation of Agglutination Titres on the Percentage of Animals Diagnosed Negative, Suspicious, or Positive for Bang's Disease

It has been shown[1] that different standards of interpretation of agglutination reactions in use in different laboratories are the cause of serious discrepancies in the diagnosis of Bang's disease. There was nearly 100 per cent disagreement in the diagnosis of 53 controversial animals (due solely to differences of opinions) by workers in nine laboratories in widely separated parts of the nation. The data presented here indicate the percentage of animals in the controversial group and point out the importance of this situation. These data were obtained in a survey[2] to determine the prevalence of Bang's disease in Minnesota.

Table I gives an analysis of the results of this survey, in which the data have been grouped in five different ways in accordance with as many different standards of interpretation of agglutination reactions. This grouping permits a study of the effect that the different standards have on the percentage of animals diagnosed in different ways. These data are not, within themselves, evidence for or against the use of any standard. They indicate the eventualities which must be dealt with when the several standards of interpretation have been used. The various standards for interpretation of agglutination titres included in table I were selected because all of them have supporters among the men working in veterinary laboratories engaged in the diagnosis and control of Bang's disease.

Complete agglutination in 1:100 dilution (+ + + — — —) is generally accepted as the condemnation level. Some workers prefer a higher titre, (at least + + + I — —) before making a positive diagnosis. These data indicate that this controversy is of only minor importance from a percentage standpoint. There

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TABLE I—*Number and percentage of animals diagnosed negative, suspicious, or positive when various standards of interpretation of agglutination titres are applied.**

WHEN	ANIMALS	
	NUMBER	PER CENT
+ I — — — — or lower = negative	5589	87.6
+ + + I — — or above = positive	476	7.5
+ + — — — — to + + + — — — = suspicious	318	5.0
Total	6383	100.1
+ I — — — — or lower = negative	5589	87.6
+ + + — — — or above = positive	554	8.7
+ + — — — — and + + I — — — = suspicious	240	3.8
Total	6383	100.1
+ — — — — — or lower = negative	5190	81.3
+ + + — — — or above = positive	554	8.7
+ I — — — — to + + I — — — = suspicious	639	10.0
Total	6383	100.0
I — — — — — or lower = negative	4564	71.5
+ + + — — — or above = positive	554	8.7
+ — — — — — to + + I — — — = suspicious	1265	19.8
Total	6383	100.0
— — — — — — only = negative	3726	58.4
+ + + — — — or above = positive	554	8.7
I — — — — — to + + I — — — = suspicious	2103	33.0
Total	6383	100.1

*Test-tube agglutination tests in dilutions of 1:25, 1:50, 1:100, 1:250, 1:500 and 1:1000.
+ = complete agglutination.
I = incomplete agglutination.
— = no agglutination or only a slight trace of agglutination.

were 554 (8.7 per cent) of the animals that showed at least a + + + I — — reaction while 476 (7.5 per cent) of all the animals showed a + + + — — — reaction. Thus, only 78 more positive reactors result from the lower (+ + + — — —) condemnation titre and these represent only 1.2 per cent of the 6,383 animals in the study. These 78 animals would be classed as suspicious if the higher titre (+ + + I — —) is selected for condemnation, and so no particular harm would result from the use of such a standard.

On the other hand, these data show that the titres selected to differentiate between negative and suspicious animals are of ex-

treme importance from a percentage standpoint. The percentage of suspicious animals varies from 3.8, when + + — — — — — and + + I — — — reactions only are classified as suspicious, to 33.0 when all reactions from I — — — — — — to + + I — — — are included in this group.

The data in the previous report[1] show that 164 (51.1 per cent) of 321 herds were classed as negative. They did not contain any suspicious or positive animals in accordance with the diagnoses as they were made. These diagnoses were not based on any fixed rules of interpretation of agglutination titres.

The data in table II show the number and percentage of these 321 herds that would be classed as negative if reactions in various low dilutions were considered suspicious of Bang's disease.

TABLE II—*Number and percentage of herds of cattle that are classed as negative when low agglutination titres are considered evidence of Bang's disease.*

WHEN	HERDS DIAGNOSED NEGATIVE AS TITRES ARE INCREASED	TOTAL	PER CENT
— — — — — — only = negative	12	12*	3.7
I — — — — — or lower = negative	26	38	11.8
+ — — — — — or lower = negative	44	82	25.6
+ I — — — — or lower = negative	68	150	46.7
+ + — — — — or lower = negative	29	179	55.8
Herds having at least one animal with a titre of + + I — — — or higher	142		
Total	321		

*Only one of these herds contained more than three animals.

These data show that 179 (55.8 per cent) of the herds would be negative if a titre higher than 1:50 is required before an animal is classed as suspicious. Probably most veterinarians would agree that this standard would not be sufficiently safe and that too many infected cows would be marked negative. Only 82 (25.6 per cent) of the herds would be negative when a titre higher than 1:25 is considered suspicious. The data show that only 82 (25.6 per cent) of these 321 herds can be classed as negative according to the recommendations of the United States Live Stock Sanitary Association as amended in 1932.[3] If all animals showing incomplete agglutination in the 1:25 dilution are considered suspicious, as was done by several laboratories in the

study previously reported,[1] there would only be 12 (3.7 per cent) of these 321 herds classed as negative. These 12 herds were mostly one- and two-cow units. In other words, these data show that there are essentially no negative herds of any considerable size in Minnesota when this standard of interpretation of agglutination titres is enforced.

II. Agglutinating Substances for Brucella Abortus Antigen in Bovine Serums Obtained from Animals Obviously Not Infected with Bang's Disease

The early, and in fact generally accepted, concept of agglutinin is expressed in the dictionary[4] definition of this term as follows:

> An antibody found in an immune serum which when added to a homogeneous suspension of its specific micro-organism causes such a change that the organisms adhere to one another and form clumps.

An antibody is defined as:

> The specific body produced by the cells of a host in reaction against an antigen; a substance in the blood and tissue juices of animals rendered immune by inoculation, and exerting a specific antagonistic influence on the substance under the influence of which it was formed.

Many authors have defined these terms somewhat differently in attempts to refine and improve the definitions and clarify their meaning. At this time, we are interested only in one phase of the discussion, namely, to present data which show that agglutinating substances for *Brucella abortus* antigen can be demonstrated in serums from animals that are not and never have been infected with Bang's disease as far as can be determined. If the cattle used in these experiments are not free from Bang's disease, we would not know where such animals might be found.

An interpretation of the data that follow is that they show that the agglutination titre or titres which are selected as a basis to differentiate between "specific" and "non-specific" agglutinins are arbitrary separations in a continuous scale of agglutination reactions extending from low to high dilutions of serum and test fluid. These separations have been made after extensive studies and experience but they are not infallible. This situation is exemplified by the fact that we have differences of opinion as to what agglutination titre should be considered evidence of Bang's disease.

The test fluid used in this experiment was prepared in accordance with the recommendations of the United States Live Stock Sanitary Association.[5] Those laboratories using other types of test fluid should not attempt to compare their results with those

reported here. The serums employed were all diagnosed negative in our laboratory, and were selected only from herds that did not contain any animals with positive agglutination titres. A total of 303 serums were tested. The tests were conducted in accordance with the above recommended technic except that a lower range of dilutions of serum and antigen was employed as follows: 1:1, 1:2, 1:5, 1:10, 1:15, 1:20 and 1:25. These dilutions were prepared by mixing appropriate amounts of undiluted serum with 1-cc amounts of test fluid.

RESULTS

All of the serums caused agglutination of the antigen in some dilution. The type of agglutination observed in these low dilutions was indistinguishable from that commonly considered as evidence of Bang's disease when observed in higher dilutions. Table III shows typical results selected at random. The maximum dilution in which agglutination was observed varied in tests of different serums.

TABLE III—*Typical results of agglutination tests in low dilutions of animals having negative diagnoses for Bang's disease.*

SERUM	DILUTIONS						
	1:1	1:2	1:5	1:10	1:15	1:20	1:25
1	+	+	+	+	+	+	I
2	+	+	+	I	—	—	—
3	+	+	+	+	I	—	—
4	+	+	+	+	+	I	—
5	+	+	—	—	—	—	—
6	+	+	I	—	—	—	—
7	+	+	+	+	+	+	+
8	+	+	+	+	+	+	—
9	+	+	+	+	+	+	I
10	+	+	+	+	I	I	—

+ = complete agglutination.
I = incomplete agglutination.
— = no agglutination or only a slight trace of agglutination.

Table IV shows the percentage of serums that did *not* cause agglutination in the various dilutions. This table indicates the number and percentage of 303 serums from non-infected animals that would have been classed as negative if these dilutions were used for diagnostic purposes and reaction in such dilutions considered as evidence of Bang's disease. For example, when a reaction in the 1:25 dilution is considered as evidence of Bang's disease, only 189 (62.4 per cent) of the 303 animals are diagnosed

negative. Or, if the 1:10 dilution had been selected, only 70 (23.1 per cent) of them would be diagnosed negative. The table is presented in the negative form, instead of indicating the number that did react in these various dilutions, because it seems more impressive to show that the percentage of negative reactions decreased to nothing when a sufficiently low dilution was employed.

TABLE IV—*Number and percentage of animals not infected with Bang's disease that do not show agglutination in low dilutions. (Total number of animals tested: 303.)*

	DILUTIONS						
	1:25	1:20	1:15	1:10	1:5	1:2	1:1
Number	189	148	115	70	28	6	0
Per cent	62.4	48.8	38.0	23.1	9.2	1.7	0

The significance of these data (table IV) is that they show that we can not consider all agglutination reactions in low dilutions as evidence of Bang's disease. An adequate explanation for the existence of "non-specific" agglutinins in the blood of non-infected cattle can not be given at this time. It is important, however, for us to appreciate the existence of such agglutinating substances even though we can not explain their presence. When an adult cow becomes infected with Brucella organisms, there is, of course, a variable latent period before she will react to the test. When this latent period has passed, specific agglutinins appear in the blood-stream in sufficient quantity so that a reaction is obtained in the dilutions used for diagnostic purposes.

The production of specific agglutinins in the animal body is more or less a gradual process. That is, the serum of the diseased animal does not show its maximum titre as soon as the first reaction is apparent. If samples of serums are collected each day from recently infected animals, usually it will be found that the first reaction noted will be in the low dilutions only, namely, 1:25 or both 1:25 and 1:50. The titre then increases to the higher dilutions. The rate of this increase is variable but usually quite rapid so that within a very few days the reaction is positive in higher dilutions. Thus a reaction in the 1:25 dilution only, on an initial test, can be indicative of a rising titre in a recently infected animal. This fact has been over-emphasized in establishing our present standards for interpretation of agglutination reactions. In formulating such standards we should

consider further facts which have been largely ignored in the present standards of interpretation.

First, it is obvious that the number of times that the serum sample will be taken during what is usually a relatively short period, when an infected animal shows a reaction in the 1:25 dilution only, is, on the law of chance, exceedingly small. Second, adequate studies[1] have shown that in excess of 20 per cent of all animals tested show a reaction in the 1:25 dilution only and that a vast majority of such cattle are not infected with Bang's disease. When we balance these two situations, we discover that the policy of placing the stigma of suspicion on all animals showing a reaction in the 1:25 dilution has the following effect. It detects a very minor percentage of animals that should be regarded as suspicious, but at the same time it disqualifies a very important percentage of non-infected healthy animals. Furthermore, we must appreciate that when the policy of considering reactions in the 1:25 dilution as evidence of Bang's disease is applied to the herd, the data (table II) show that there are essentially no negative herds of cattle. This is, of course, contrary to fact as borne out by the clinical evidence in many herds.

White and his associates[6] have reported extensive studies pertaining to standards of interpretation of agglutination results and have arrived at similar conclusions regarding the significance of reactions in the 1:25 dilution. The experience of many workers engaged in the study and control of Bang's disease has taught them that it is unwise to consider all reactions in the 1:25 dilution as evidence of Bang's disease. Consequently, as indicated in a previous report,[1] we find that many laboratories are not complying with the standard for interpretation of agglutination results as recommended by the United States Live Stock Sanitary Association. We recommend that this standard be modified.

III. A Schematic Representation of the Overlapping Ranges of "Non-Specific" and "Specific" Agglutination Reactions

The fundamental principle emphasized in the preceding data is not new. The fact that some cases must be classified as suspicious, since we are not justified in diagnosing them as either negative or positive, has been recognized for years. A similar situation exists in all biological tests for the diagnosis of disease. We can all recall many more or less unsatisfactory experiences in tuberculosis control work that were due to the suspicious cases.

These data add to our knowledge of the situation involving suspicious cases in Bang's disease by indicating the percentage of

animals that are found in the different categories. While it is advisable to recognize that we have the problem of separation of "non-specific" and "specific" reactions so that we may use this method of diagnosis more intelligently, it is of paramount importance for us to guard against overemphasis of this problem. The graph in table V illustrates the problem. The preceding data show that the problem does not assume an important rôle in the whole program of control of Bang's disease except when we ignore the facts and establish unreasonable standards for interpretation of agglutination results. The overlapping zones of agglutination illustrated in the graph do not involve an important number of cattle when the agglutination results are interpreted in accordance with the facts. These data, along with other data referred to here, teach us that we must show some consideration for the non-infected animal that carries "non-specific" agglutinins when conducting and interpreting the agglutination test. When this is not done, the percentage of suspicious cases becomes too large to be dealt with in an acceptable manner, and too many non-infected cattle are classified as suspicious.

TABLE V—*Overlapping zones of specific and non-specific agglutinations.*

DILUTIONS OF SERUM IN TEST FLUID								
1:1	1:5	1:10	1:25	1:50	1:100	1:250	1:500	1:1000
Range of "non-specific" agglutination ——————?								
							Range of "specific" agglutination ?——————	

The explanation of the graph in table V is as follows: Practically all bovine serums contain agglutinating substances for *Brucella abortus* which can be demonstrated by using a sufficiently low dilution of serum and test fluid, say 1:1. Some serums contain such substances in larger quantity than others, or at least in a form that causes agglutination in higher dilutions, whether this variable be quantity or quality of agglutinating substances. The maximum dilution in which agglutination will be observed in testing serums from "non-infected" cows is variable and is indicated with a question mark in the graph. The question mark is used to indicate that we do not feel competent to determine its upper limit for all cases. It is placed higher than the 1:25 dilution be-

cause we feel sure that most reactions in the 1:25 dilution only are not evidence of Bang's disease.

Likewise, in the graph, the line representing the range of "specific" agglutination begins at 1:1000. All animals whose serums agglutinate the antigen in this dilution should be considered infected. This is a safe policy whether or not the bacteria can be isolated from all such cows. This line ("specific" agglutination) terminates with a question mark, because the lower limit is not a fixed value. It terminates, however, at a point lower than the upper limit of the line representing the range of "non-specific" agglutination. Thus we have an overlapping of the two ranges, probably somewhere between the limits of about 1:25 and 1:100.

Some investigators may criticize this graphic representation, because it does not include any provision for pro-agglutinoid zone reactions. We have previously shown[7] that many observations of this phenomenon are due to an unsatisfactory test fluid (*e. g.*, using formalin as a preservative). Very few pro-agglutinoid zone reactions are observed with the test fluid as recommended by the United States Live Stock Sanitary Association. When such reactions are observed, they are possibly frequently due to some unsatisfactory chemical property of the serum-antigen mixture and not necessarily to properties of the serum agglutinins themselves. This is supported by the observation that pro-agglutinoid zone reactions are rarely, if ever, observed in the rapid agglutination test.[8] Jones and Orcutt[9] have recently presented an excellent report on this subject in which they seem to have shown that pro-agglutinoid zone reactions in two bovine serums were due to an inhibitory substance in the serum which could be separated from the agglutinin in the serum.

Discussion

These data emphasize two important points pertaining to the control of Bang's disease. First, the effect, from a percentage standpoint, of a lack of uniformity in standards of interpretation of the agglutination test. Second, the recognized fact that one of our problems in the control of Bang's disease is to differentiate between "non-specific" and "specific" agglutination reactions. There may be at least two possibilities for accomplishing this objective. One would be to devise a method of agglutination testing which would inhibit or obliterate "non-specific" reactions without affecting "specific" reactions. Such a method is not available. The other possibility is in use at the present time, namely, to establish arbitrary separations between "non-specific" and "specific" agglutination titres. These data may be used to

establish standards of interpretation of results on a sounder basis. It seems clear, however, as has long been recognized, that no standard will be infallible. Most men recognize that there are a very few infected animals that do not react even in low dilutions. Also, that there are some cows that show a reaction in the lower part of the diagnostic range of dilutions from which *Brucella abortus* can not be isolated and with no evidence that such cows are actively infected. We should fix our standard of interpretation so that a minimum number of diseased cattle will pass the test and at the same time guard against the evil of condemning too many non-infected, non-dangerous animals.

While standards of interpretation are needed as a guide, they do not need to be and should not be inflexible. The existing chaotic condition regarding the interpretation of agglutination results is clearly shown in these data. This situation is definitely the result of attempts to evolve a fixed standard of interpretation.

The necessity for using a fixed standard of interpretation of agglutination reactions can be largely avoided by basing diagnoses of suspicious cases on a consideration of the complete status of the entire herd rather than only on the agglutination titre of the individual animal. For example, we would prefer to accept, as non-infected, a cow showing a low agglutination titre (say 1:25) from a herd not containing any animals having definitely positive reactors than to accept a cow having a completely negative reaction from a badly diseased herd. In making diagnoses we prefer to make use of all available information pertaining to the entire herd, such as the age of the animal, the breeding history, stage of gestation and all clinical evidences of disease. It should be remembered, however, that the uncertainty in diagnosis of individual animals is confined to the suspicious group, and this group is only a minor one according to reasonable standards of interpretation of agglutination results. These data show that when all animals showing agglutination in the 1:25 dilution only are considered suspicious, this group then comprises about one-third of the total.

In the case of animals intended for interstate shipment, the practice of requiring that all animals in the herd be tested no doubt would meet with opposition in some quarters. However, it is the only really sound method of making diagnoses of Bang's disease. We should consider the agglutination test as an invaluable aid in making a diagnosis of Bang's disease in the suspicious cases, but should not consider it the sole source of evidence. Diagnoses made in accordance with hard and fast rules for interpretation of agglutination results have ignored this fundamental

principle of diagnosis, and this policy has resulted in the chaotic situation exemplified in these data. In spite of some opposition to this policy, due to some slight inconvenience to cattle-buyers intending to ship animals interstate, at least one state has already incorporated this principle of diagnosis in its regulations pertaining to Bang's disease. This state requires that all animals in the herd must be certified to by the sanitary officials of the state of origin as being free from Bang's disease before any animal in the herd will be accepted as disease-free. Such regulations may be too severe at the present time. They are basically sound, however, and the policy of requiring tests of all the animals in the herd before making diagnoses of individuals showing low agglutination titres is a step in the right direction.

SUMMARY

1. Data are given which indicate the effect, from a percentage standpoint, of different standards for interpretation of agglutination results.

2. Data are presented which disprove the idea that all agglutinating substances demonstrated in low dilutions should be considered as evidence of active infection with *Brucella abortus*.

3. The inadequacy of inflexible standards for interpretation of agglutination results is emphasized by these data. The policy of making diagnoses of suspicious cases in Bang's disease only after careful consideration of the complete status of the entire herd is advocated.

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FURTHER STUDIES ON TRANSMISSION OF EQUINE ENCEPHALOMYELITIS BY MOSQUITOES*

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Since the early work of Kelser,[1, 2] continued interest has been manifest in problems pertaining to mosquito transmission of equine encephalomyelitis.

The authors[3-5] have reported their results on the transmission of equine encephalomyelitis from guinea pig to guinea pig by means of two species of mosquitoes native to Utah. As reported, most of the attempts at transmission were negative. Therefore, little information regarding the period of infectivity of the mosquito was obtained. Attempts were made also to infect various lots of mosquitoes by feeding them on 50 naturally infected horses during the 1934 epizoötic. All gave negative results. Such mosquitoes were later either re-fed on healthy guinea pigs or were emulsified and injected intracranially into guinea pigs; many of the horses fed upon were in the height of their febrile reaction. Experience from such experimentation prompted a study to determine at what period of the disease the insects are capable of obtaining virus from the peripheral circulating blood and over what periods these mosquitoes remain effective transmitters.

METHODS

Adult mosquitoes were captured in insect nets near margins of swampy areas in Cache and Boxelder counties. Certain locations were found to yield practically pure cultures of either *Aedes dorsalis* or *Aedes nigromaculis*, the species caught depending upon the time of year and the area visited. These were brought to the laboratory in gauze-covered cages, 14x14x24 inches. These cages then were placed in a specially constructed humidor which maintained a constant temperature of about 62° F. The humidor was approximately 5 feet square and 7 feet high and was constructed from two thicknesses of burlap tacked on a frame. The burlap was dipped into a shallow pan that rested on the top, which was kept filled and slightly overflowing with water by means of a hose connected to a water-tap. The mosquitoes were given their infective meal upon infected guinea pigs, which were rather snugly

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tied into a piece of 21-mesh gauze; these were either placed on top of the mosquito cage or were suspended in the gauze bag against the sides of the cage by means of thumb tacks. Excellent feedings were obtained by this method, as many as several thousand mosquitoes being fed upon four guinea pigs at a given interval.

From three to five infected guinea pigs were used to provide each infective feeding for the mosquitoes; the same series of guinea pigs often were used for several feedings at various intervals. The fed mosquitoes were then removed to somewhat smaller cages, and during the time that transmission refeeding tests were carried out, 50 mosquitoes were removed daily to several small celluloid cylinders. The gauze ends of these cylinders were then placed against the clipped body of the animal to be fed upon. An average of 35 mosquitoes were later re-fed on each healthy guinea pig, after which the mosquitoes were placed in a "residue" cage. When all mosquitoes in the original cage had been used (usually 12 to 16 days following the infective feeding), the mosquitoes in the residue cage were again re-fed. By this method it was possible to get feedings with as long an interval as 25 days between infective and transmission feedings. One group of mosquitoes usually was re-fed on the third or fourth day following infective feeding, and transmission feedings continued each day with a fresh group until no vigorous mosquitoes remained.

Guinea pigs to be used for infective feedings of mosquitoes were infected by introducing, between the two cerebri, 0.2 cc of a 2 per cent emulsion of brain tissue containing virus. All infected guinea pigs used in this work were temperatured and weighed daily until they succumbed. The same weighing and temperaturing procedure was followed with the transmission-test guinea pigs, except that in a portion of the trials weights and temperatures were taken only on alternate days. If the pigs showed no obvious symptoms at the end of the 15 days following mosquito feeding, recording of weights and temperatures was discontinued.

Two sources of western type of virus were used: (1) Virus C was used in most of the trials. It was recovered in July, 1934, and is the same virus which was shown capable of mosquito adaptation in the earlier report.[4] (2) A virus recovered from the Idaho 1935 outbreak through the courtesy of Dr. W. T. Huffman was used in only a limited number of trials and with negative results in mosquito transmission attempts. This virus produced symptoms in intracranially injected guinea pigs which were similar to symptoms produced by virus C. Positive and negative re-

sults were based on the rapid weight loss, 3° to 6° F. increase in temperature, the rather characteristic cat pose, and later paralytic symptoms. Some cases only partially fulfilled these requirements; for the purpose of clarity in tabulation, they were regarded as negative transmission tests. In a few positive and suspicious cases the brain was emulsified and inoculated into susceptible guinea pigs as a further check on the presence of the virus. In the mosquito emulsion test, suspensions were prepared by emulsifying 15 mosquitoes in 2 cc of buffered saline solution and allowed to stand in the refrigerator from one to three hours, after which 0.2 cc was injected intracranially.

Discussion

A study of tables I and II shows a rather low incidence of transmission, only 5.8 per cent of the total trials with *A. nigromaculis* and 2.5 per cent of the total trials using A. *dorsalis* being positive. Data indicate that, among guinea pigs, *A. nigromaculis* is a more consistent transmitter. This may be due to its more vigorous feeding habits or it may provide greater virus multiplication or greater ability to release virus into the host. It is worthy of note that in this study the general period of infectivity in the two species differs (table III). In *A. nigromaculis* the period varied between four and ten days, while in *A. dorsalis* the period varied between nine and 19 days. The most fertile incubation period for *A. nigromaculis* was six, seven and eight days. The few positive cases when using *A. dorsalis* were more or less scattered, making it difficult to determine the most consistent period of transmission; however, 12, 13 and 14 days would probably be the most likely choice.

Using monkeys in mosquito transmission experiments with yellow fever, Hudson and Philip[6] found a direct relationship between the febrile reaction of the donor animals and the virus incubation period within the mosquitoes.

Mosquitoes which became infective at the height of donor febrile reaction, and presumably when the virus was at its greatest concentration in the blood, proved infective after an incubation period of ten and twelve days, whereas mosquitoes fed during the pre-febrile and post-febrile periods were not infective at 13 and ten days but became transmitters at 25 and 20 days. Such a correlation was not clearly evident in our work with equine encephalomyelitis, although the tendency may be suggested to a slight degree, inasmuch as *A. nigromaculis* showed the greatest incubation period of ten days on the 60th hour of donor infectivity. Only one transmission was recorded beyond this hour, *viz.*,

TABLE I—*Summary of data using Aedes nigromaculis; 275 guinea pigs were used in transmission attempts with 16 positive transmissions.*

Days	Hours from Virus Injection to Infective Feeding of Mosquitoes										
	12	18	24	30	36	42	48	54	60	66	72
3	N*	N	NN	N		N	N				N
4	NN	NNN	NNNN	NNN	N	NNN	NP†	NN	N	N	N
5	NN	NNNN	NNN	NNN	NN	NNN	P	N	N	N	N
6	NN	NNNN	NNNNP	NNNP	NP	NNN	N	NN	N	N	N
7	NN	NNN	NNNP	NNP	N	NN	N	N	N	N	N
8	NN	NP	NPP	NNP	NP	NNN	N	NP	N	P	N
9	NN	NP	NNNN	NNN	N	N	N		N	N	N
10	NN	NN	NN	NNN	N	NNN	N	NN	P		N
11	NN	NNN	NNN	NN		N		N			N
12	N	NN	NN	NNN	N	NN	N	N	N	N	N
13	N	NN	NN	NN	N	NN	N	N	N	N	
14	N	N	N	NN	N	NN	N	N	N		N
15	N	NN	NN	N		NN				N	N
16	N	N	N	NN		N	NN	NN			N
17	N		N	NN	N	N			N	N	
18	N	N	NN	N	N	NN	N	NN			
19		N		N	NN	N				N	
20			N	N		N			N		
21		N		N	N						N
22			N			N					N
23					N						
24			N	N							
25											
Mosquito emulsion tests (6 days)	NNP	NNNP	NPPP	NNNNP	NN	NNPP	P	NP	P	P	N

*Each N represents one guinea pig with negative results.
†Each P represents one guinea pig with positive results.

†Each P represents one guinea pig with positive results.

TABLE II—*Summary of data using Aedes dorsalis; 244 guinea pigs were used in transmission attempts with 6 positive transmissions.*

Days	Hours from Virus Injection to Infective Feeding of Mosquitoes										
	12	18	24	30	36	42	48	54	60	66	72
3	N*	NN	NNN	N	NN	NN	NNNN				
4	N	NN	NNNN	NN	NN	NNN	NNNN	NN	N	N	N
5	N	NN	NNN	N	NN	NN	NN	N	N	N	N
6	N	NN	NNNN	NN	NN	NNN	NNN	NN	N	N	N
7	N	NN	NNN	N	NN	NN	NNN	N	N	N	NN
8	N	NN	NNN	NN	N	NN	NN	NN	N	N	N
9		N	NN	N	NP†	NN	NNN	N	N	N	N
10	N	NN	NN	NN	NN	NNN	N	NN	N	N	NN
11	N	NN	NN	N	NN	NN	N	N	N	N	N
12	N	NP	NN	NN	NN	NNN	N	NN	N	N	N
13	N	NN	NN	N	NP	P	N	N	N	N	N
14	N	N	N	N	NN	NNP	N	NN	N	N	N
15	N	N	N		N	N	N	N	N	N	N
16	N	N	N	N		N		N	N	N	N
17		N			N	NN		N			
19				N	N	P					
20	N	N						N			
24								N	N	N	N
Mosquito emulsion tests (6 days)	P	PPP	PPP	NP	PPPPP	PPPPPN	PPP	NN	P	N	N

*Each N represents one guinea pig with negative results.
†Each P represents one guinea pig with positive results.

TABLE III—*Periods at which mosquitoes were transmitters: Aedes nigromaculus, 18 to 66 hours following donor infection; A. dorsalis, 18 to 42 hours following donor infection. (Data within lines shown in tables I and II.)*

	Days Following Transmission Feeding														
	4	5	6	7	8	9	10	11	12	13	14	15	16	17	19
A. nigromaculis (Percentage of trials positive)	5	5	13	12	39	7	7								
A. dorsalis (Percentage of trials positive)						12.5	0	0	9	25	12.5	0	0	0	33

the 66th hour. *A. dorsalis* showed the greatest incubation period of 19 days on the 42nd hour of donor infectivity, beyond which no transmissions were recorded. It would seem from experiments conducted by the authors with equine encephalomyelitis that the greatest virus availability period for mosquitoes occurred in the pre-febrile stage. Intracranially-infected guinea pigs usually showed their peak febrile reaction between 66 and 96 hours. Thus, there appeared to be a slight tendency for the longer incubation transmissions to occur from feedings made following the peak of virus availability. Merrill *et al.*[7] found that when mosquitoes were fed on infected guinea pigs they did not become transmitters of equine encephalomyelitis unless engorgement took place when the blood showed a high titre of virus (first febrile reaction), even though the mosquito was capable of virus multiplication. In investigations conducted by the authors, *A. nigromaculis* was capable of obtaining active virus from the donor, from 18 to 66 hours following donor infection (table IV). From casual examination of table I, it would appear that the most fertile period was 24 and 30 hours. However, when it is considered that more guinea pig trials were made in this period than in others, it follows that the positive cases were just as frequent in the later periods, except for the 42nd hour (which showed no transmissions). Using the data between incubation periods of four and ten days, inclusive, the values in percentage of transmissions were obtained as shown in table IV for *A. nigromaculis*. The same data from the ninth to the 19th, inclusive, are figured for *A. dorsalis*.

It is not clear why *A. dorsalis* failed to transmit later than the 42-hour feeding (table IV), whereas *A. nigromaculis* continued to be infective up to and including the 66-hour feeding; it may be due, however, to experimental variation, since the positive cases

TABLE IV—*Periods at which the native mosquitoes obtained virus from donor guinea pigs, as indicated by subsequent transmission to healthy guinea pigs. (Data within lines shown in tables I and II.)*

	Hours Following Donor Infection									
	18	24	30	36	42	48	54	60	66	72
A. nigromaculis (Percentage of trials positive)	10	16	14	20	0	25	10	14	17	0
A. dorsalis (Percentage of trials positive)	8	0	0	13	16	0	0	0	0	0

in the study were infrequent with *A. dorsalis*. In a number of attempts to demonstrate virus in the circulating blood of intracranially-infected guinea pigs, animals of 24-hour duration showed virus more consistently than did those of 48- and 77-hour duration. The total mortality rate of 5.8 per cent for *A. nigromaculis* and 2.5 per cent for *A. dorsalis* is not necessarily a measure of the fatalities of truly exposed animals, since many of the guinea pig trials apparently were made outside the periods of conceivable transmission. If we determine the percentage of transmission within the box rules drawn on tables I and II, it is observed that there was 12.7 per cent of fatalities using *A. nigromaculis* and 8.6 per cent fatalities using *A. dorsalis*.

These mortalities do not differ greatly from the morbidity rate of horses in Utah-infected areas reported by Madsen in 1934. In this report the morbidity during the first outbreak was 3.8 per cent, while in the second outbreak the morbidity rate was 6.9 per cent. It is conceivable that some of the guinea pigs used in the transmission experiments encountered some virus invasion without visible symptoms of disease. The temperature curve of a few pigs suggested such a possibility, although as a group those pigs reported as negative showed no temperature curve.

Table V gives a summary of transmission attempts using horses, supplemented with guinea pigs. In these attempts at transmission the recently recovered Idaho virus was used. The horses purchased were from ranches which presumably had not encountered the disease, although all horses were from communities which had been invaded by the disease during the previous two years. Horse 2, which was bitten by approximately 500 infective mosquitoes (*A. dorsalis* and *A. nigromaculis*), twelve days following their infective meal obtained from diseased guinea pigs, was found prostrate 31 days following such mosquito exposure. Other than the prostration and icteric condition of the visible mucous

TABLE V—*Summary of mosquito transmission attempts using horses, supplemented with guinea pigs, using Aedes nigromaculis and Aedes dorsalis.*

Animals Providing Infective Feeding	Interval Between Animal Inoculation and Infective Feeding of Mosquitoes (hours)	Interval Between Infective Feeding of Mosquitoes and Transmission Feeding (days)	Animal Used in Transmission Feedings	Insects Fed	Transmission Results	Mosquito Emulsion Tests
Horse 6	20 to 23	4	G. P. 812	30	Negative	
Horse 6		4	G. P. 551	36	Negative	
Horse 6		7 to 9	Horse 4	1000	Negative	Negative
Horse 6		10 to 14	Horse 5	300	Negative	Negative
Horse 6	30 to 36	4	G. P. 942	40	?	
Guinea pigs		6	G. P. 432	50	Negative	Positive
Guinea pigs		6	G. P. 872	25	Negative	Positive
Horse 6		7 to 9	Horse 4	1000	Negative	
Horse 6		10 to 14	Horse 5	300	Negative	Negative
Guinea pigs		12	Horse 1	725	Negative	Negative
Guinea pigs		12	Horse 2	500	Negative	Negative
Guinea pigs		15	G. P. 751	25	Negative	Negative
Guinea pigs		15	G. P. 865	27	Negative	Negative
Guinea pigs		17	G. P. 890	32	Negative	
Guinea pigs		17	G. P. 557	33	Negative	Negative
Guinea pigs		18	Horse 3	90	Negative	Negative
Guinea pigs		23	G. P. 322	15	Negative	

membranes, there was no special evidence of encephalomyelitis. Autopsy revealed a general icterus. Repeated attempts to recover virus from the brain and anterior cord were negative. The animal was aged and was quite emaciated when purchased, and it is believed that death was incident to old age. Temperatures taken over this period did not show any rise above normal. Blood from horse 4 was tested daily (except the 10th) for virus content, with negative results from the second to the 13th day following mosquito exposure. Horses 1 and 2 were negative on the second and third days following mosquito exposure. Horses 1, 3 and 5 were temperatured and observed for 40 days following mosquito exposure, after which they were exposed to active virus. Horses 1 and 5 were exposed by intracranial injection of virus. Horses 3 and 4 were exposed by intralingual injections of virus; horse 5 was fatally injured in restraint apparatus during the operation. The remaining three horses proved capable of resisting the disease and showed no evidence of increased temperature or other symptoms. The virus introduced to the horses killed control guinea pigs in the characteristic manner. Unfortunately, blood serum from these horses was not tested for specific virus-neutralizing substances prior to mosquito exposure. It is evident that the horses harbored some type of neutralizing substance in their bodies subsequent to mosquito exposure. This protective mechanism could either have been conveyed through field exposure one or two years previous to the test or it might have developed following mosquito exposure. Epizoölogical studies have shown that a considerable number of presumably exposed horses never show clinical symptoms. This observation suggests that mosquito exposure in the field may induce mild subclinical infections which confer immunity.

Horse 6, which provided the infective feeding for a number of mosquito groups, was infected by intracranial infection of active virus. This horse subsequently showed a characteristic course of the disease (paralytic type) and became prostrate; it was destroyed on the fifth day. Virus could not be demonstrated in the blood of this horse at the 21st, 45th, 54th, and 66th hours following injection, and subsequent tests did not reveal active virus in the brain and anterior cord.

No explanation is offered as to why investigations with native mosquitoes did not result in more frequent transmission. Virus concentration in the circulating blood, virus multiplication within the mosquito, the ability of the mosquito to transfer this virus to a new host, the neurotropic, viscerotropic and hemotropic predi-

lections of individual viruses are all factors which require further elaboration.

SUMMARY

The period of transmissibility of equine encephalomyelitis virus in *Aedes nigromaculis* varied between four and ten days, with the peak at six, seven, and eight days. In *Aedes dorsalis* the period varied between nine and 19 days, with twelve, 13, and 14 days as the most likely choice of a peak. The period of virus availability in the donor for *A. nigromaculis* was between 18 and 66 hours, inclusive. In *A. dorsalis* the period was from 18 to 42 hours, inclusive. A limited number of attempts to transmit the disease to horses by means of these two native mosquitoes were unsuccessful.

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Milk and Water

A rather unusual story appeared in the June 20, 1936, issue of *Today* under the title, "Milk and Water," by George Weller. The thing that makes the story unusual is the fact that a veterinarian is one of the leading characters. The story deals with cows, the tuberculin test, indemnities for reactors, and crooked milk distributors. The latter were responsible for the water mentioned in the title. *Today* is edited by Raymond Moley.

PERSONALS

DR. MARTIN D. PEARL (U. P. '34) has moved from Philadelphia, Pa., to Atlantic City, N. J.

DR. MICHAEL D. DUCEY (Ont. '16), recently of Detroit, Mich., has removed to Midland, Mich.

DR. J. L. O'NEILL (O. S. U. '36), of Troy, Ohio, has chosen Sidney, same state, to establish a practice.

DR. K. S. JONES (O. S. U. '36), formerly of Rushsylvania, Ohio, has opened an office at Kenton, same state.

NOTES ON THE PREPARATION AND USE OF AN ANTIGEN FOR THE AGGLUTINATION TEST IN SWINE ERYSIPELAS*

By A. W. DEEM, *Columbus, Ohio*
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The agglutination test for the serological diagnosis of swine erysipelas, using *Erysipelothrix rhusiopathiae,* presents two difficulties: (a) growing the organisms in sufficient quantities to produce an antigen; and (b) preparing an antigen that stays in suspension and does not show spontaneous flocculation.

Schoening, Creech and Gray,[1] of the Pathological Division, Bureau of Animal Industry, U. S. Department of Agriculture, use an agglutination test which gives very satisfactory results, but has not been reduced to the simplicity of the test for Bang's disease in cattle. For growing the organisms they use a standard beef infusion broth, which, while reasonably satisfactory, gives a rather scanty yield. To overcome the effect of spontaneous flocculation or settling out of the bacteria, they incubate the tests for only 30 minutes and then centrifuge for 4 minutes at 1,800 revolutions, at which time the reading is made.

We have attempted to overcome or lessen both these difficulties. Many different kinds of media, both liquid and solid, were used. We tried the addition of many enrichment substances to standard media, most of which were unsatisfactory either because the growth was not appreciably increased or, as more often occurred, because they tended to increase non-specific flocculation or sedimentation. This we believe is due largely to some types of media favoring dissociation, or converting smooth to rough forms. We have never been able to prepare a satisfactory antigen from a typically R type in which the organisms grow in long filaments.

By experimentation we found that the addition of tryptophane and dried brewer's yeast to beef infusion broth greatly accelerated the growth, with a minimum of spontaneous flocculation. We have since been using this medium for growing the organisms for our antigens. It is prepared as follows: to each 1,000 cc of meat juice (1 pound lean beef soaked overnight in 1 liter of water and juice pressed out) are added 7.5 gm commercial peptone, 2.5 gm "Difco" Dehydrated Tryptophane broth and from 0.5 to 1 gm dried brewer's yeast. This is heated to boiling and adjusted to pH 7.2. After heating again to boiling, it is filtered through cotton and then through paper. Sterilization is done

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at 15 lbs. pressure for 20 minutes. To make filtering easier the yeast may be added in a small cheese-cloth bag. The bacteria are grown in 500-cc Florence flasks, each containing 350 to 400 cc of broth, during incubation for 48 hours at 37° C. The result is a very good and quite uniformly turbid growth.

Our next attempt was to prepare an antigen which would definitely stay in suspension. Although unable to use the full yield of organisms grown, the antigen developed stays in suspension and has given good results with known positive sera. It is prepared by growing the organisms in the beef infusion yeast broth for 48 hours. This is centrifuged for one hour at 2,000 r.p.m. and the supernatant fluid discarded. The sedimented bacteria are next suspended in 95 per cent alcohol for 18 to 24 hours, which seems to minimize flocculation. The organisms are again concentrated by centrifuging for one hour at 2,000 r.p.m. and the supernatant fluid discarded. The packed organisms are resuspended in 0.9 per cent salt solution containing 0.5 per cent phenol, only enough diluting fluid being used to give a rather heavy bacterial suspension. This is put in the shaking-machine for one hour without beads. We then centrifuge again for ten minutes at 1,000 r.p.m. This throws down bacterial clumps and filaments which would otherwise settle out on standing and interfere with the reading of the tests. Only the supernatant suspension is saved and diluted, if too concentrated, to the density of tube 1 on the McFarland nephelometer scale, after which it is ready for use.

In conducting the test, 1 cc of antigen is used in each tube, adding to the respective tubes the proper amounts of serum and incubating for 24 hours at 37° C. The test is then ready to be read.

The antigen control and negative serum tubes show little or no sedimentation, and we have immune sera which gives a complete agglutination in dilutions of 1:2,000.

Summary

Beef infusion broth containing yeast and tryptophane provides an excellent medium for the growth of *Erysipelothrix rhusiopathiae* as the antigen in the agglutination test in swine erysipelas.

A method is described for the preparation of an antigen that stays in suspension so that the test may be read after incubation for 24 hours.

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THE EFFECT OF BRUCELLA ABORTUS INFECTION OF THE UDDER ON THE QUALITY OF MILK*

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Brucella abortus may invade the bovine udder and cause a low-grade mastitis. The work of Sholl and Torrey[1] demonstrates that *Br. abortus* produces definite changes in the udder that may be rather extensive and may lower the efficiency of the organ considerably. The mastitis is usually not sufficiently extensive to result in an alteration of the physical appearance of the milk secreted.

Results of work of this Station on the quality of milk produced by streptococcus-infected udders have suggested that possibly the invasion of the udder by *Br. abortus* might result in changes in the milk that could be detected by bacteriological and chemical tests designed to measure the quality of milk, even though its physical appearance remained unchanged. If any such changes in quality of milk are incident to *Br. abortus* infection, it is expected that they would not be so great as those found in streptococcic mastitis, where frequently the physical appearance of the milk is greatly altered. This study was undertaken to determine what effect *Br. abortus* infection of the udder has on the quality of milk and to compare the results with those obtained upon examination of the milk from healthy udders and udders affected with streptococcic mastitis.

EXPERIMENTAL METHODS

The cows of two herds were placed at the disposal of the writers insofar as this study was concerned. Fourteen and 13 cows were located in herds A and B, respectively, with *Br. abortus* infection of the udder and no streptococcic mastitis. The presence of *Br. abortus* infection of the udder was detected by cultural and serological examination of the milk. Only cows yielding the organism on culture at periodic examinations, either with or without a positive agglutination test of the milk serum, were included in this study. In addition, the microscopic test for streptococcic mastitis[2] was run on all milk samples; only cows negative to this test were studied.

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Composite and quarter samples of milk from each cow were aseptically collected and examined according to standard methods for the tests employed. The methylene-blue reduction time and bacteria count were determined on the composite samples. Our aim was to simulate practical dairy farm conditions where the milk of such cows is used irrespective of *Br. abortus* infection, since the physical appearance of the milk usually is not altered. The leukocyte content, percentage of chlorides, and pH (thybromol test) determinations were made on the quarter samples and if found abnormal in any one quarter the cow's milk was considered abnormal by that test. This gave the true picture of conditions present, whereas dilution by milk from the negative quarters would make such chemical tests of composite samples inaccurate.

RESULTS AND DISCUSSION

Milk samples were collected periodically and examined from the cows selected; these cows had only *Br. abortus* infection of the udder. The study was continued for a period of nine months in herd A and for six months in herd B. A few individual cows both ended and began lactation periods during the study; this accounts for the variation in the number of animals examined each time. In all cases, samples were taken only from the animals originally selected for the study. The several cows that subsequently became affected with streptococcic mastitis were no longer included in this study, and records on them were not considered; therefore, the changes reported are assumed to be due to *Br. abortus* infection.

The data are grouped according to the total number of cows examined at each sampling period. The records for individual cows are not given, since it would unnecessarily enlarge the material presented; actually the variations obtained are indicated in the total number of samples occurring in the various classes of the tests employed, at each sampling. The results from herd A and herd B are presented separately, so as more clearly to indicate the condition found in each herd. These data are presented in table I.

Of the 118 samples from herd A examined by the methylene-blue reduction test, 87.2 per cent were class 1, 8.4 per cent class 2, 2.5 per cent class 3, and 1.9 per cent class 4, as compared to the 46 samples from herd B where 74 per cent were class 1, 19.4 per cent class 2, 4.4 per cent class 3, and 2.2 per cent class 4. The milk from the *Br. abortus*-infected cows in herd B was not so high in quality as that from the cows in herd A, as meas-

TABLE I—*Results of examination of milk from cows with Brucella infection of the udder without streptococcus infection.*

HERD	DATE	COWS	COW COMPOSITE SAMPLES									QUARTER SAMPLES*							
			METHYLENE-BLUE CLASS				BACTERIA PER CC					LEUKOCYTES PER CC			CHLORIDES		THYBROMOL		
			1	2	3	4	LESS THAN 100	100 TO 200	200 TO 500	500 TO 1000	MORE THAN 1000	LESS THAN 500	500 TO 1000	MORE THAN 1000	LESS THAN 0.16%	MORE THAN 0.16%	—	±	+
A	7–16–35	14	14	0	0	0	3	2	2	0	7	13	1	0	14	0	12	0	2
	7–20–35	14	13	1	0	0	6	2	2	0	4	13	1	0	13	1	12	0	2
	7–25–35	14	14	0	0	0	4	3	2	0	5	13	1	0	13	1	11	0	3
	8– 2–35	14	13	1	0	0	0	2	3	2	7	13	1	0	9	5	11	0	3
	8–12–35	14	13	1	0	0	0	1	4	3	6	12	2	0	4	10	10	2	2
	9–25–35	12	10	2	0	0	0	3	4	3	2	12	0	0	10	2	9	1	2
	10–15–35	8	6	1	1	0	1	2	1	2	2	7	0	1	8	0	3	1	4
	11–19–35	8	6	1	0	1	0	4	3	0	1	7	0	1	6	2	5	1	2
	12–27–35	11	7	2	2	0	1	2	2	2	4	8	2	1	5	6	6	0	5
	3– 3–36	9	7	1	0	1	1	2	1	3	2	9	0	0	9	0	7	1	1
	Totals	118	103	10	3	2	16	23	24	15	40	107	8	3	91	27	86	6	26
	Samples (%)		87.2	8.4	2.5	1.9	13.5	20.0	20.0	12.7	33.8	90.7	6.8	2.5	77.0	23.0	72.8	5.0	22.2
B	10–29–35	13	10	2	1	0	4	5	2	1	1	11	2	0	12	1	9	1	3
	11– 8–35	13	8	4	1	0	3	5	2	2	1	10	3	0	9	4	9	2	2
	12–11–35	9	8	1	0	0	0	3	3	1	2	6	2	1	7	2	8	0	1
	3– 3–36	11	8	2	0	1	1	4	2	2	2	11	0	0	10	1	9	0	2
	Totals	46	34	9	2	1	8	17	9	6	6	38	7	1	38	8	35	4	8
	Samples (%)		74.0	19.4	4.4	2.2	17.3	37.2	19.5	13.0	13.0	82.6	15.3	2.1	82.7	17.3	73.9	8.7	17.4

*If udder is abnormal in any quarter, cow is abnormal.

ured by the methylene-blue reduction test. The significant observation is that a high percentage of the samples are in classes 2, 3 and 4.

The bacteria count of the milk samples varied from one sampling to the next. As indicated in table I, in herd A, 66.2 per cent of the samples contained less than 1,000 bacteria per cubic centimeter and 33.8 per cent over 1,000, as compared with herd B, where 87 per cent of the samples contained less than 1,000 per cc and 13 per cent a count of greater than 1,000. Just as the count varied among cattle of the same herd, it varied in the two herds.

The samples were further classified according to the number of leukocytes per cc as determined by Breed's technic. In a study of the relation of streptococcus infection to udder induration, Hucker and Udall[3] set the normal value at 500,000 or less per cc. Other workers[2, 4] recognize the normal as 1,000,000 or less leukocytes per cc of milk. The samples examined are classified according to both of these standards.

The leukocyte content of the milk samples indicates that a low-grade mastitis was present in the cows with *Br. abortus* infection of the udder. Of the total number examined in herd A, 90.7 per cent, and in herd B, 82.6 per cent contained less than 500,000 per cc and according to Hucker and Udall this would indicate the absence of udder induration. In addition, in herd A, 6.8 per cent contained more than 500,000 and less than 1,000,000, and 2.5 per cent of the samples contained more than 1,000,000 leukocytes per cc. Similarly, 15.3 per cent of the samples of herd B contained between 500,000 and 1,000,000 and 2.1 per cent more than 1,000,000 leukocytes per cc of milk. The classification of samples of the two herds was almost identical.

The normal value of 0.16 per cent chlorides as given by Hucker[3] was used as the basis of our classification. One of the common changes produced by mastitis is an increase in the chloride content of milk. In herd A, 77 per cent of the samples did not contain excess amounts of chlorides, while 23 per cent were higher than normal. The records for herd B were similar, for 82.7 per cent of the samples did not contain excess amounts of chlorides, while 17.3 per cent had increased amounts of chlorides. These figures indicate that a low-grade mastitis was present in the cows with *Br. abortus* infection of the udder.

The thybromol test is a color reaction to determine the pH of milk, using brom thymol blue as an indicator. Negative (—) indicates a pH of 6.6 or less, suspicious (±) refers to a pH

of 6.7 to 7.0, and positive (+) a pH of greater than 7. The pH of milk samples from both herds corresponded, since in herd A, 72.8 per cent of the samples were negative, 5.0 per cent suspicious, and 22.2 per cent positive, and in herd B, 73.9 per cent were negative, 8.7 per cent suspicious and 17.4 per cent positive. These data confirm the work of Morrison and Hull,[6] in which they found that 20 samples of milk, from a series of 132 from positive cows, or approximately 16 per cent, reacted to the thybromol test. The data reported by Fitch and Bishop[7] also agree with the above; they report that the milk from 13 out of 34 *Br. abortus*-infected quarters (26 per cent) gave a positive thybromol reaction.

In order to indicate the relationship of *Br. abortus* infection of the udder and quality of milk produced to the quality of milk from healthy and that from cows affected with streptococcic mastitis, the data of table II are presented. The figures for quality of milk from healthy cows, and those with streptococcic mastitis are taken from the work of Bryan and Trout,[8] and unpublished data on the same series of cows. The figures of herds A and B in table I are averaged to obtain the value for the *Br. abortus*-infected cows.

When classified by the methylene-blue reduction test, 98.5 per cent of the samples from the healthy udders were class 1, 80.5 per cent of the *Br. abortus*-infected milk samples were class 1, and only 52.6 per cent of the samples from streptococcus infected udders were class 1. On this basis, the healthy udders produced milk of highest quality, the streptococcus-infected udders produced milk of lowest quality, and the *Br. abortus*-infected udders produced a better milk than the streptococcus-infected udders, yet much decreased in quality as compared to the milk from healthy udders.

The bacteria counts of the composite samples are also significant. It is valuable to know the exact count of milk, yet in the production of high-quality milk importance is attached only to counts exceeding 1,000 bacteria per cc. Only 5.8 per cent of the disease-free udders produced milk with a count of more than 1,000, as compared with 23.4 per cent of the *Br. abortus*-infected, and 68.5 per cent of those affected with streptococcic mastitis. The *Br. abortus* infection did not increase the count as much as the streptococcus infection, yet the count was greatly increased as compared with normal.

The leukocyte count gives some information concerning the activity of the infection in the udder. The streptococcus infec-

TABLE II—*Results of examination of milk produced by cows with udder infection and with no udder infection. The data give the percentages of samples occurring in the various classes of the tests employed.*

Infection Involving Udder	Cow Composite Samples									Quarter Samples*							
	Methylene-Blue Class				Bacteria per cc					Leukocytes per cc			Chlorides		Thybromol		
	1	2	3	4	Less Than 100	100 to 200	200 to 500	500 to 1000	More Than 1000	Less Than 500	500 to 1000	More Than 1000	Less Than 0.16%	More Than 0.16%	—	±	+
None	98.5	1.5	0	0	14.4	7.7	26.3	45.8	5.8	95.0	4.3	0.7	90.0	10.0	88.4	7.6	4.0
Brucella only	80.5	14.0	3.4	2.0	15.4	28.5	19.8	12.9	23.4	86.7	11.0	2.3	79.8	20.2	74.5	6.9	19.8
Streptococci only	52.6	28.0	15.9	3.5	0	0	7.0	24.5	68.5	23.0	27.0	50.0	27.0	73.0	34.5	17.4	47.8

*If udder is abnormal in any quarter, cow is abnormal.

tion causes the greatest increase and *Br. abortus* infection a slight increase in leukocytes per cc of milk, as indicated by the fact that 50 per cent of the streptococcus-infected samples, 2.3 per cent of the *Br. abortus*-infected samples and 0.7 per cent of the normal samples contained more than 1,000,000 leukocytes per cc of milk. In fact, the milk samples from healthy cows with leukocytes in excess of 1,000,000 per cc were obtained from cows near the end of their lactation period.

The comparative study of data on chloride content again presents similar results. In other words, 10 per cent of the samples from healthy udders contain abnormal amounts of chlorides as compared with 20.2 per cent of the samples from *Br. abortus*-infected udders and 73 per cent of the streptococcus-infected udders. These data indicate that *Br. abortus* infection of the udder does alter the chloride content of the milk but not so markedly as does streptococcus infection of the udder.

It is significant that in the thybromol test 19.8 per cent of the *Br. abortus*-infected udders produced milk of abnormal pH as compared with 47.8 per cent of the streptococcus-infected udders and 4.0 per cent of the healthy udders. In making pH determinations of quarter samples of milk, it is essential to bear in mind the part that *Br. abortus* infection of the udder may play.

SUMMARY

The quality of milk secreted by *Br. abortus*-infected udders is decreased in quality as compared with that from healthy udders, but the changes are not so pronounced as those found in streptococcic mastitis.

Br. abortus may cause a low-grade mastitis, as has been indicated by previous histopathological studies and by the results of the tests employed in this study.

The chemical composition of milk may be altered as a result of *Br. abortus* infection of the udder.

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PAPILLARY ADENOMA OF THE GALL-BLADDER IN TWO DOGS: INTRAHEPATIC GALL-BLADDER IN ONE*

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Papillary adenoma of the gall-bladder of the dog, especially with extensive involvement of the cystic duct, is an unusual finding. The discovery of two such cases seems to warrant their report. Of additional interest is the intrahepatic situation of the gall-bladder in one of these cases.

Nieberle and Cohrs[1] stated that adenoma of the gall-bladder is not infrequent in dogs. Feldman[2] did not report any cases of papillary adenoma of the gall-bladder. He[3] stated that our two cases were the first he had seen. Reports of similar cases could not be found in the American veterinary literature.

The occurrence of similar growths in man is not frequent. Marshall[4] could find, in the literature, reports of only twelve cases of benign papilloma of the bile-ducts in man. They may occur in the gall-bladder or in any portion of the extrahepatic biliary passages. It is considered that these growths usually arise from the surface epithelium and that they are potentially malignant.

Report of a case of an intrahepatic gall-bladder in a dog could not be found in the literature. Mentzer,[5] in an extensive study of the comparative anatomy of the biliary system of more than 100 animals seen in Africa, found none with an intrahepatic gall-bladder. Halpert[6] has reported the finding of an intrahepatic gall-bladder in a Rhesus monkey and in the pig. A few more than 25 cases of intrahepatic gall-bladder in man have been reported. It is possible that many of the cases of so-called congenital absence of the gall-bladder are cases of true intrahepatic gall-bladder. It is difficult to explain the reason for this situation of the gall-bladder but it is undoubtedly because of some error

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in development. It is thought that the gall-bladder is buried in the liver in the course of normal development and undoubtedly in some cases the gall-bladder persists in this position in adult life.

Reports of Cases

Case 1: A female police dog, 13 years of age, in excellent general condition, was killed by inhalation of ether. Necropsy was performed immediately. The presence of a well-formed gall-bladder notch, but complete absence of the organ itself, was noted. In the middle of the anterior surface of the right central lobe of the liver was a bluish area, about 3 cm in diameter, which appeared as if a cyst lay beneath it. The cyst-like process was

Fig. 1. Case 1. Longitudinal section of the right central lobe of the liver, showing the gall-bladder completely surrounded by hepatic tissue, the empty gall-bladder notch, the cavity of the gall-bladder containing sandy material, and the papillary growth filling the neck of the gall-bladder.

thought to be the fundus of the gall-bladder. It was tense and conformed to the regular contour of the hepatic surface. The entire liver was removed. A gall-bladder could not be found on the dorsal surface. A cystic duct, of normal appearance, protruded from the substance of the liver and joined the extrabiliary system in the usual situation.

The entire liver was cut through on a longitudinal plane. A gall-bladder, somewhat larger and with thicker walls than usual, was found completely surrounded by the substance of the right central lobe of the liver (fig. 1). The bladder was filled with a thick, green type of bile, characteristic of stasis. There was an

enormous amount of sandy material but no true gall-stones were found. Completely filling the neck and extending throughout the cystic duct, but not into the common bile-duct, was a friable, papillary growth. The cystic duct appeared to be almost completely obstructed.

Microscopically, the liver was normal, with the exception of the cells adjacent to the gall-bladder. The wall of the gall-blad-

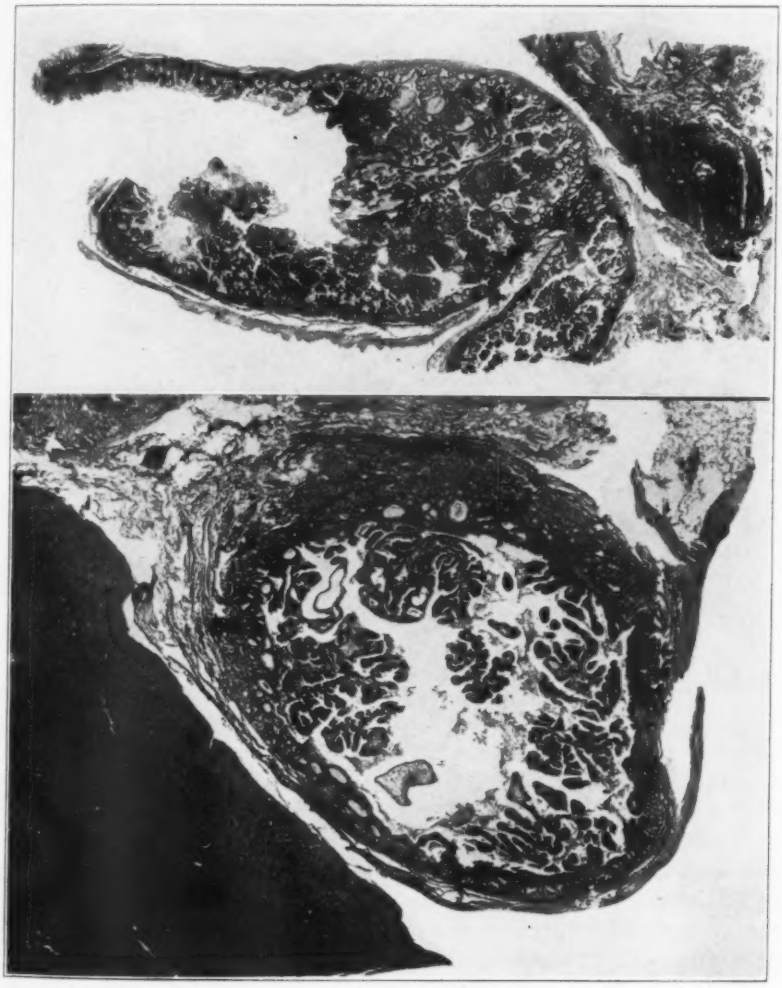

FIG. 2 (above). Case 1. The neck of the gall-bladder and cystic duct, showing the papillary growth (x 4).

FIG. 3 (below). Case 1. Cross section of the cystic duct at its junction with the extrahepatic biliary system, showing the papillary growth (x 11).

der was complete and the number of lymphoid cells within it was increased. There was a marked, intricate piling up of papillary outgrowths of the mucosal epithelium of the neck of the gall-bladder and of the entire cystic duct (figs. 2 and 3). This was growing into the lumen and appeared very hyperplastic. In

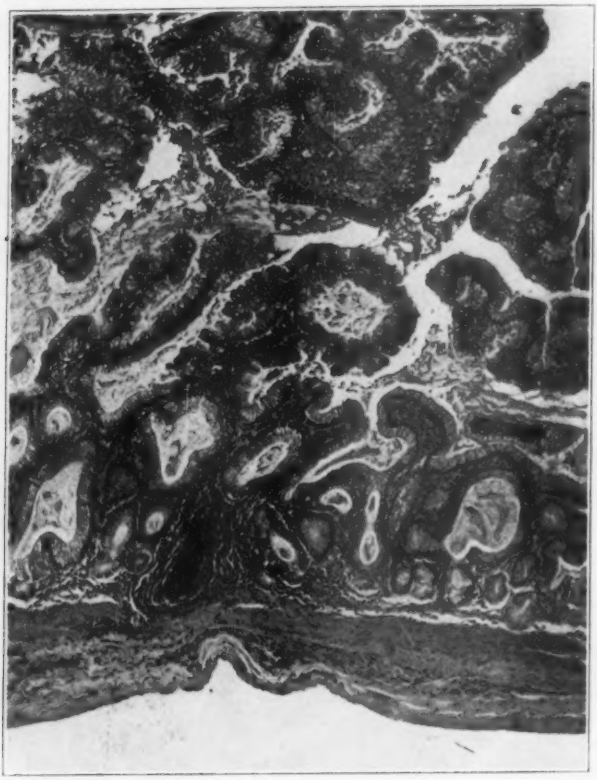

FIG. 4. Case 1. Section through the wall of the gall-bladder, showing the papillary mucosal outgrowths. The mucosal epithelium is of hyperplastic but benign appearance and the lymphoid cells in the wall of the gall-bladder are increased (x 70).

one area the wall of the gall-bladder was invaded by regular, papillary outgrowths but in no instance did the overgrowths of the cells extend through the wall of the gall-bladder or cystic duct (fig. 4). The cells were of tall columnar type and in none was mitosis seen. The mucosa was covered with a large amount of mucus. Some of the more pedunculated areas of the growth gave evidence of necrosis but no signs of infection were present.

In many of the epithelial cells retrogression was evidenced by pyknosis and karyorrhexis. There was evidence throughout the stroma of yellowish crystals which were probably bile salts.

Case 2: A well nourished, female fox terrier, twelve years of age, was destroyed, at the owner's request, by inhalation of ether. Necropsy was performed immediately. Bilateral corneal opacity had caused nearly complete blindness. The following pathologic condition of the liver and gall-bladder was observed: The liver was of normal size and color, but grossly it had the appearance characteristic of biliary cirrhosis. The gall-bladder had a mottled appearance and was distended and tense. The cystic duct was about twice normal size. The common bile-duct was normal. After fixation in 10 per cent formalin solution, the gall-bladder

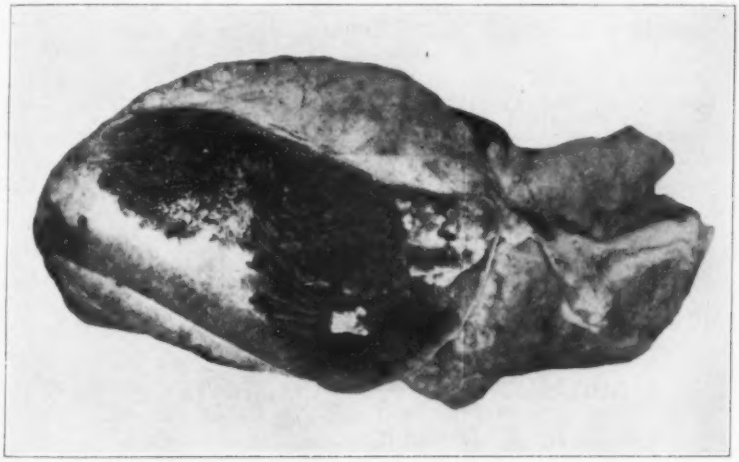

FIG. 5. Case 2. Longitudinal section of the gall-bladder and cystic duct, showing the cavity partially filled with a papillary growth arising in the neck. There is sandy material in the fundus.

and cystic duct were opened longitudinally. The gall-bladder was filled with a type of bile characteristic of stasis and numerous sandy particles. There were no true gall-stones. Arising in the neck and extending well into the cavity of the gall-bladder was a friable, soft wavy, villous-like mass (fig. 5). This fungating growth also extended throughout the proximal two-thirds of the cystic duct.

Microscopically, the liver appeared normal. The gall-bladder and growth were almost duplicates of those in case 1. However, the growth in case 2 was less hyperplastic and did not appear to be growing as rapidly.

Comment and Summary

Two cases of benign, papillary adenoma of the gall-bladder of the dog, with extension into the cystic duct, are presented. In one there was an associated intrahepatic gall-bladder. The absence of symptoms of disease of the gall-bladder in both cases is of interest. The partial occlusion of the cystic duct, with the resulting stasis, probably accounts for the presence of the sandy material in the gall-bladders of both dogs. It is highly probable that if these papillary adenomas, which were very hyperplastic and rapidly growing, and which involved the wall of the gall-bladder, had been allowed to progress, they might eventually have shown the characteristics of true malignancy. They must be classified, at least, as potentially malignant growths. It is interesting to correlate these findings with those seen in man, because they so closely parallel the findings in man.

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A METHOD OF TREATING ENTROPION*

By E. J. Frick, *Manhattan, Kan.*
Department of Surgery and Medicine, Kansas State College

The following procedure has worked satisfactorily on one colt, one calf and nine dogs, four of which were Chows, suffering from various degrees of entropion. Although this method does not entirely supplant the radical surgical operation, it should be tried first, as it will frequently do away with the necessity of operating.

The eye to be treated is first carefully examined to see if only one lid is involved and about how extensive the inversion has occurred. The head of the animal should be held firmly and the skin of the lid wiped clean. Using a sterile, glass 5-cc syringe, filled with sterile, heavy mineral oil, and with an 18-gauge needle, insert the needle parallel to the lid margin and inject a

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FIG. 1. Making subcutaneous injection of oil to correct entropion.

varying amount of oil subcutaneously under the lid until the lid bulges out to the normal position and the entropion is corrected. (See figure 1.)

A little practice will soon tell how much each particular case needs. If you do not use enough oil, the lid may turn in again and the injection will have to be repeated at a later date. The advantages of this method are that it does away with the surgery and stitch irritations that have so many complications. It gives immediate relief and can be completed in a few minutes. It leaves no scar.

The disadvantages are that in a very extensive entropion the operation will not give permanent results and in order to be successful a little intelligent practice is required to judge the placing and the amount of oil to be injected. Perhaps some other cosmetic material, such as paraffin wax, would be better than oil.

TETANUS IN A HEIFER, WITH SPONTANEOUS RECOVERY*

By W. S. O'NEAL, *Saint Charles, Mo.*

Subject: Yearling, grade Jersey heifer.

History: No history of injury or illness of any kind. Never bred. Always in fair to good condition. No ear-tag.

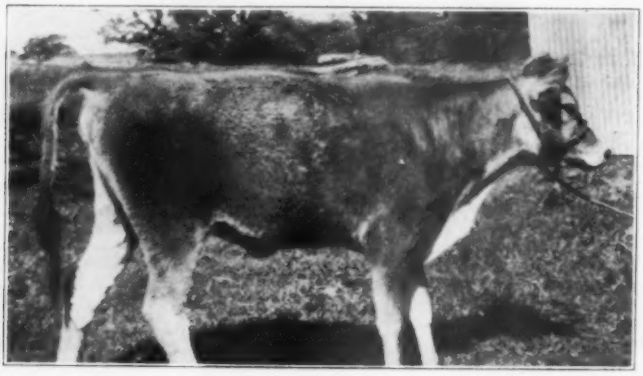

FIG. 1. Heifer showing symptoms of tetanus.

Symptoms: Head and neck extended; tail extended; ears erect; musculature in tetanic contraction. (See figure 1.) Moderate tympany of rumen; reflex irritability increased; stiff, straddling

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gait; marked, but not complete, trismus of masseter muscles. Protrusion of membrana nictitans, with characteristic "jerky" movement of tetanus.

Examination: Careful examination failed to reveal the slightest evidence of trauma.

Diagnosis: Tetanus.

Prognosis: Unfavorable.

Treatment: None. The heifer was allowed to run in pasture as usual.

Recovery: After one week, the symptoms began to subside and in three weeks the heifer had made a complete recovery.

PARASITISM AND TUBERCULOSIS IN A CROW*

By F. R. BEAUDETTE *and* C. B. HUDSON
New Jersey Agricultural Experiment Station
New Brunswick, N. J.

On January 2, 1936, Mr. Leslie Black brought in two crows which he had shot on a farm near Stockton, Hunterdon County, New Jersey.

The postmortem examination showed the presence of gizzard worms and tapeworms in each bird. The gizzard worms were thought to be *Acuaria anthuris* and were later positively identified as such by E. E. Wehr. It might be added that this is a common parasite of crows in this section.

Blood-smears were made of each bird and stained with Wright's stain. The examination showed many microfilaria in one of the specimens. These larvae were sheathed.

Mr. Black was requested to bring in additional crows and a week later was fortunate in picking up what was thought to be a crow with an injured wing. An examination of the blood was made as soon as the bird was removed from a darkened box in the morning and microfilaria were found but no sheath was seen.

This bird was kept in the laboratory until it died, on June 12. During this interval, several examinations were made in the daytime but microfilaria were never found. Suspecting that the larvae appeared only at night, a few examinations were made after 9:00 p. m., and in every instance larvae were plentiful.

These larval forms exhibited unusual viability, as evidenced by the fact that they were still active after four days under cover-slips sealed with vaseline and held at room temperature.

*Journal Series paper of the New Jersey Agricultural Experiment Station, Department of Poultry Husbandry. Received for publication, June 29, 1936.

A search of the literature revealed that Elliott[1] found microfilaria in the blood of three of eleven crows in Canada in 1901. On this occasion he failed to find the adults. However, one of four crows examined the following year showed the same parasite and, upon careful dissection, the adults were found in the coats of the pulmonary artery. Elliott indicated that he would make a further report on the adults but apparently this was never made.

According to Dr. Cram,[2] *Diplotriaena tricuspis*, which is found in the body-cavity, is the only adult filarid reported in crows in this country, but no one has associated the larval forms with an adult. I am indebted to Dr. Cram for the following references to the literature which show that microfilaria have been reported in crows, in 1845, by Gros[3] and Ecker,[4, 5]; in 1852, by Herbst[6]; in 1891, by Linstow[7] and in 1933, by Markowski.[8]

When the crow died, a careful search was made for the adult forms, especially in the wall of the pulmonary artery, but none could be found. A blood-smear revealed microfilaria more abundant than had ever been seen during life. About 4 cc of water was placed in each body-cavity and aspirated into a tube which was centrifugalized. An examination of the sediment revealed a myriad of larval forms. These may have come from the blood but the number found would suggest that they might have been present otherwise. Curiously enough, the larvae exhibited no motility although the carcass was still warm.

There was a small area of necrosis on one border of the liver. Numerous caseous nodules were found in each lung and throughout the kidneys. A few millet seed-sized nodules were loosely attached to the right peritoneal wall. A very large nodule was attached to the left cecum. The center of this had broken down to leave a cavity communicating with the gut.

The right carpal joint was considerably enlarged and appeared to be fractured. However, on opening it, the joint was found to be entirely destroyed and contained a quantity of caseous material. Smears made of this as well as from the liver and nodules from the kidney showed numerous acid-fast organisms.

Tuberculosis in crows has already been reported by Mitchell and Duthie,[9] who found five infected out of 36 from western Ontario, and one infected out of four from Ottawa.

Tuberculosis in poultry is not a common disease in New Jersey. Thus, in 13,306 autopsies between 1923-1928, only 22 cases were found[10] and they have been equally rare since then.

We wish to express our appreciation to Mr. L. Black, for the specimens, and to Dr. Cram, for references to the literature.

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REPORT OF A PENILE ULCER IN A DOG*

By CARL F. SCHLOTTHAUER *and* LEONARD K. STALKER
The Mayo Foundation, Rochester, Minn.

Penile ulceration is not a common finding in a dog. A case with an associated hematuria that caused diagnostic difficulties was thought to be of sufficient interest to report.

REPORT OF A CASE

A male fox terrier, aged 18 months, was brought to one of us (Schlotthauer) because of an illness which had been present for six days. Six days previous to our examination, the dog had a cough, purulent nasal discharge, lassitude and anorexia. The owner had administered two worm capsules. Since this time, there had been hematuria. Examination revealed a very sick and dyspneic animal with a temperature of 104.6° F. There was a marked conjunctivitis, pharyngitis and a nasal discharge. Palpation of the abdomen did not reveal any abnormality. There was a constant dribbling of what appeared to be bloody urine. Palpation of the urinary bladder revealed it to be empty and therefore a catheter was not inserted. The diagnosis of distemper with pneumonia and a probable nephritis was made.

The animal was given 10 cc of anti-canine distemper serum and was treated for the nephritis at home. He became progressively worse and on the fifth day of his illness was again brought to us for observation and further treatment. At this visit, 12 cc of distemper serum was administered. There was no improve-

*Received for publication, June 22, 1936.

ment and the animal died on the 17th day after the onset of the disease.

Necropsy revealed bilateral bronchopneumonia. Gross and microscopic examination of the kidneys, bladder and urethra did not reveal any abnormality. There was a small punched-out ulcer, 1 cm in diameter, situated in the middle of the dorsum of the glans penis (fig. 1). This was an acute process with little surrounding inflammation. The ulcer reached a depth of about 2 to 3 mm. There was no evidence of trauma of the penis or of the surrounding organs. The urethra was patent and appeared normal. The postmortem diagnosis was distemper, bilateral bronchopneumonia, and bleeding of the penis.

FIG. 1. Ulcer of the glans penis of a dog.

COMMENT

We feel certain that the hematuria in this case was the result of the penile ulceration. It was probably a very minor complication, as the dog died as a result of distemper and pneumonia, but a nephritis, which was originally suspected and for which treatment was instituted, would have been a serious complication. We hope that the report of this case and the error made in diagnosis, as a result of incomplete examination of the genitourinary apparatus, will stimulate others to make a careful examination of the external genitalia as well as of the urinary tract

in all cases of hematuria. We are unable to explain the cause of the penile ulceration in this dog, but suspect that its origin may have been traumatic with secondary infection.

SUMMARY

We have presented a case in which penile ulceration in a dog caused hematuria with an associated distemper.

NAPHTHALENE POISONING IN POULTRY*

By C. B. HUDSON, *New Brunswick, N. J.*

On September 5, 1934, a poultryman presented a hen and a pullet for examination. According to the owner, out of a population of 400 birds, 18 were lost in three weeks and others in the group were sick. The first symptom noticed in the affected birds was the development of a greenish black diarrhea. Death invariably followed within three days. Among another population of 900 birds on the same premises, similar losses were not encountered.

The symptoms observed in the birds presented were identical. Their combs were red and their eyes bright. There was a partial paralysis of the legs. The feathers around the vent were soiled with greenish black fecal material.

On autopsy the hen showed the following changes: There were small blister-like areas in the mucosa of the crop. The liver was swollen and showed small necrotic areas. The intestines were catarrhal and the gizzard contents gave off a strong odor of naphthalene. A moth-ball was found in the crop.

In the case of the pullet, a moth-ball was found in the crop, and the gizzard contents gave off a strong odor of naphthalene. The gall-bladder was distended and the intestines were catarrhal.

After the finding of the moth-balls, the owner stated that he had placed them in the nests in order to protect the birds against lice and mites. However, no moth-balls were placed in the nest boxes of the 900 birds in which there had been no losses.

Later it was learned from the owner that shortly after the moth-balls were removed from the nests, all losses stopped. However, 40 birds were lost, all showing identical symptoms, as described above.

*Journal Series paper of the New Jersey Agricultural Experiment Station, Department of Poultry Husbandry. Received for publication, July 3, 1936.

ANOMALOUS URINARY SYSTEM IN A SMALL PIG*

By J. F. BULLARD, *LaFayette, Ind.*
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Purdue University Agricultural Experiment Station

The subject in this case was a small, eight-week-old Chester White male pig that weighed approximately 20 pounds. It had

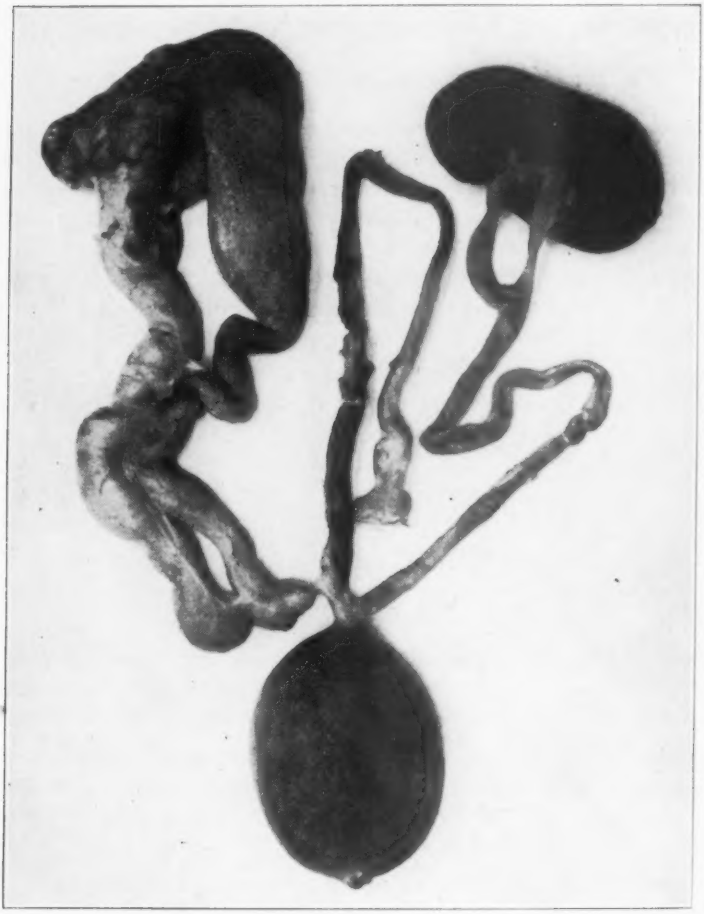


FIG. 1. Shows an anomalous urinary system taken from a small pig. The abnormal ureters are shown to unite directly with the urethra.

been on experiment and when this was terminated the pig was culled and destroyed on account of being unthrifty.

*Received for publication, July 13, 1936.

At autopsy no outstanding inflammatory processes were seen. The main lesions were confined to the urinary system. A large, convoluted tubular mass occupied the major portion of the right side of the abdominal cavity. It appeared to replace the right kidney and extended posteriorly into the pelvic cavity.

The left kidney was apparently the only one that was functional. It was slightly enlarged for a pig of this size; which, in all probability, was due to a compensatory hypertrophy. It had what seemed to be two ureters emerging from the hilus.

All other abdominal viscera were carefully removed before the urinary system was dissected. A detailed dissection revealed several very interesting anomalies. The right kidney was very cystic and from its anterior extremity originated two tubular structures, each 2.5 cm in width. Five cm from their origin, they united to form one common ureter which was 19 cm long. From the posterior extremity one single, greatly dilated ureter was given off which was 4 cm wide and 9 cm long. After this it became greatly constricted, measuring only 1 cm in width. It continued for 17 cm in this form and was held in close contact to the ureter from the anterior extremity by dense connective tissue strands. These two united to form one common ureter 2 cm long, which became constricted rather abruptly. This portion of the ureter was 0.5 cm long and 2 mm wide and, instead of discharging into the bladder, was in common union with the urethra, 2 cm from its origin at the neck of the bladder.

From the hilus of the left kidney there originated two ureters, 3 cm from each other. Each was 0.5 cm wide and 4.5 and 5 cm in length, respectively. They united and formed one common tortuous ureter 27 cm long. It in turn united with the urethra 0.5 cm from the neck of the bladder. All passages were patent in both the right and left ureters.

The bladder was moderately distended with a thin, greenish yellow, purulent, urine.

Figure 1 shows the relationship of these structures. For convenience in photographing, in order to conserve space, the kidneys and ureters, instead of being above the bladder, were turned down parallel with the urethra and penis. The right ureters were dissected and separated in two places in order to show their relationship.

English Teacher: "William, what are pauses?"
Bill: "Them's things what grows on dogs."

—*Animaldom.*

THE INFLUENCE OF THE LIVER IN THE FORMATION AND DESTRUCTION OF BILE SALTS. Jesse L. Bollman and Frank C. Mann. Amer. Jour. Physiol., cxvi (1936), p. 24.

The normal dog excretes but traces of bile salts in the urine, and bile salts are not definitely detectable in normal blood. Intravenously injected glycocholates and taurocholates rapidly disappear from the blood and only a small portion appears in the kidneys. After complete removal of the liver, bile salts are not found in the blood or urine even after periods of time in which they are readily detected following ligation of the common bile-duct. The liver appears to be the site of formation of bile salts. After complete removal of the liver, injected bile salts are readily excreted in the urine so that quantitative recovery of the injected material is usually made within twelve hours. If similar injections are made in animals with biliary obstruction, only part of the injected material can be recovered. The liver apparently plays a predominant part in the destruction of bile salts. Formation of bile salts is inhibited by administration of hepatotoxins such as chloroform, carbon tetrachloride or tetrachlorethane, but is not affected by other toxins such as toluylenediamine. Destruction of bile salts is little altered by hepatotoxins that markedly alter the formation of bile salts.

LESIONS IN THE PANCREAS AND IN THE ANTERIOR HYPOPHYSIS WITH FATAL ACIDOSIS FOLLOWING PROLONGED INTRAVENOUS ADMINISTRATION OF GLUCOSE IN DOGS. Henry R. Jacobs and Arthur R. Colwell. Amer. Jour. Physiol., cxvi (1936), p. 194.

Prolonged and continuous administration of large amounts of glucose by vein to normal dogs resulted in: (1) early and sustained improvement in the capacity to make use of the administered sugar, failing terminally; (2) remarkable hepatic storage of glycogen; (3) progressive depletion of the alkali reserve, resulting ultimately in fatal acidosis; (4) generalized mild congestive changes in all viscera; (5) specific intense hemorrhage into

and destruction of the pancreas and the anterior lobe of the hypophysis. The authors surmise the formation of excessive amounts of an unidentified acidic intermediate product of glucose metabolism.

A Preliminary Report on the Injurious Effect of Sodium Carbonate in Chicks. J. F. Witter. Poultry Sci., xv (1936), p. 256.

Sodium carbonate in drinking water for poultry has been commonly used in enteric conditions. This compound in the usual dosage (¼ pound to 5 gallons of water) caused chicks to drink more water than normal and produce moist droppings. Chicks two weeks old developed pale and enlarged kidneys but chicks three weeks of age or older showed no noticeable injury. Chicks two to eight weeks of age were seriously injured within one to three days and some deaths occurred when twice the above amount of sodium carbonate was added to the drinking water. Similar results are reported when four times the original dose was placed in the drinking water. Kidneys from chicks affected by sodium carbonate showed increase in weight, paleness, engorgement with urates, and degenerative and exudative changes. The author suggests that if sodium carbonate is used in the drinking water, the dosage must be gauged accurately to satisfy the age tolerance of the chicks.

Cattle Plague Vaccine. Studies on glycerinized spleen pulp. S. C. J. Bennett. Jour. Comp. Path. & Therap., xlix (1936), p. 1.

Virulent cattle plague tissues treated with twice their weight of 60 per cent glycerin become avirulent in three months at 0° C. and in three days at 37° C. Glycerinized tissues are as safe as those treated with formalin or toluene. The vaccine, regardless of size of dose, produces a serviceable immunity in all cattle and a solid immunity in most cattle within three days. After a week, it produces a solid immunity in all cattle. Glycerinized vaccine retains its antigenic value when stored in the cold for longer periods than when stored at higher temperatures. The duration of immunity in the herd is ten to twelve months. The size of the dose does not have a great relative influence on duration of immunity. A serviceable immunity was not conferred by less than 0.5 gm per 100 kg of spleen or 0.2 gm per kg of lymphatic gland.

INORGANIC PHOSPHORUS AND PEROSIS. John C. Hammond. Poultry Sci., xv (1936), p. 260.

Studies were made of the correlation between the percentage of chicks showing perosis in each lot and percentage of total phosphorus, inorganic phosphorus, organic phosphorus and calcium in the diet. Data on 191 lots of Rhode Island Red chickens indicated correlation between inorganic phosphorus and total phosphorus and perosis in the order named and a negative correlation between perosis and organic phosphorus. Inorganic phosphorus is a primary factor in the etiology of perosis. There is a factor in rice bran which assists in the regulation of calcium and phosphorus metabolism in chickens.

BORDER PINING IN SHEEP. W. Lyle Stewart and A. Phyllis Ponsfield. Jour. Comp. Path. & Therap., xlix (1936), p. 49.

Analyses of pastures on three farms in Northumberland on which "pining" in sheep occurred suggested deficiency in calcium, phosphorus and iron, one or all three minerals being involved in varying degrees and possibly inducing specific malnutrition. The net energy value of the grazing was sufficient for maintenance if the sheep succeeded in obtaining an adequate daily intake, but the sparse distribution of the readily eaten herbage may have been possibly responsible for general undernutrition at certain times of the year. Nematode parasites belonging chiefly to the subfamily, *Trichostrongylineae*, were shown to be the apparent cause of the main symptoms of acute pining, but the numbers of parasites present during the winter months were insufficient to account for the persistent anemia and emaciation characteristic of chronic pining. Border pining is no doubt due to a combination or interaction of malnutrition and gastrointestinal parasitic infestation.

TRANSMISSION EXPERIMENTS WITH BOVINE MALIGNANT CATARRH. R. Daubney and J. C. Hudson. Jour. Comp. Path. & Therap., xlix (1936), p. 63.

Two strains of malignant catarrh from Kenya proved to be transmissible. The first strain was maintained in two passages in cattle experimentally and corresponded clinically and pathologically to Goetze's "mild" form of the disease. The "mild" strain is irregularly transmissible by blood inoculation, usually after a long incubation period. The more virulent "head and eye"

form is transmissible by blood, brain and gland inoculation, with uniform regularity and an exceedingly high mortality. The lack of uniformity between different strains has probably caused the divergence of opinions held by various workers on the subject. The experimentally produced disease has not been readily transmissible by contact. The disease has been transferred from cattle to rabbits. In early rabbit passages the lesions in the kidney, liver and brain are identical with those in cattle. In later passages the infective agent appears to become increasingly neurotropic with a corresponding reduction in the severity of the visceral lesions. A fifth passage of an intraperitoneal strain which has retained the viscerotropic character has been maintained. The disease has been transferred from rabbits to cattle in typical fatal form and to sheep in a mild form.

FURTHER STUDIES ON THE EFFICACY OF FOWL-POX VACCINE. A. Komarov and I. J. Kligler. Jour. Comp. Path. & Therap., xlix (1936), p. 90.

Fowl-pox vaccine applied by the stick method on the wing, to flocks two to three months old of early hatch, results in a few secondary lesions and confers a durable immunity. It is not advisable to use this vaccine in late-hatched birds because those birds suffer from severe post-vaccination reactions. It is suggested tentatively that late-hatched birds be first vaccinated with pigeon-pox vaccine and two months later with fowl-pox.

VACCINATION AGAINST TUBERCULOSIS, A COMPARATIVE STUDY IN MAN AND ANIMALS. H. J. Corper, A. P. Damerow, M. L. Cohn and C. B. Vidal. Jour. Inf. Dis., lviii (1936), p. 158.

It was demonstrated that in animals there is a retardation of virulent tuberculous infection resulting from a previous vaccination with viable avirulent human and bovine tubercle bacilli. Avirulent tubercle bacilli in amounts exceeding 0.001 mg of fine suspension per cc produce in man and animals definite intracutaneous local lesions similar to those resulting from the infection of the same non-viable tubercle bacilli. Avirulent human or bovine (BCG) tubercle bacilli do not produce progressive lesions when injected intracutaneously in man, when injected in amounts not causing ulceration (0.01 mg); they lose their viability in these lesions within six months. There exists a biological specificity of the immune reaction in animals with avirulent

bovine tubercle bacilli against virulent bovine infection which would suggest a greater efficiency from the use of avirulent human tubercle bacilli against virulent human infection. When viable, avirulent tubercle bacilli are to be used for vaccinating, it would appear advisable to use intracutaneous injections for this purpose and to use an amount of bacilli well within the range of producing visible reaction and yet not excessive to the point of producing abscesses or ulcers. Two or more semi-monthly or monthly intervals would serve to reinforce the desired effect.

THE SEROLOGICAL CLASSIFICATION OF THE BRUCELLA GROUP. Lyle Veazie and K. F. Meyer. Jour. Inf. Dis., lviii (1936), p. 280.

By means of monospecific serums, over 400 strains of Brucella from 20 countries were classified. All smooth cultures were easily separated into two main types corresponding to the serologic "abortus suis" and the "melitensis" types. A small subtype was observed, consisting of 26 strains which was similar to the abortus type in antigenic makeup. It is differentiated from the true abortus type by a greater proportion of melitensis antigen. A comparison of these findings with dye reactions and hydrogen sulfid production revealed conflicting results in only 5.8 per cent of the tests. Ten per cent of the cultures identified as "bovis" types by dye reactions are serologically indistinguishable from melitensis cultures. All of the cultures classified as "suis" type by biochemical reactions fall into the abortus type antigenically. Forty-two per cent of these strains were isolated from a single herd of cattle in the United States. Their reaction indicates the value of systematic classification in epidemiological studies. The failure of differentiation tests in a certain percentage of cases serves to emphasize the fallacy of relying on any one test for proper and complete identification.

A DIFFERENTIAL STUDY OF FORTY BRUCELLA STRAINS ISOLATED IN MINNESOTA. Paul Kalber and Margaret MacLanahan. Jour. Inf. Dis., lviii (1936), p. 293.

Forty Minnesota strains of Brucella were classified as follows: 25 strains *Brucella suis*, 13 strains *Brucella abortus* and two strains with conflicting reactions but one appeared to be *Brucella suis* and the other *Brucella melitensis*. The agglutinin-absorption method is of little or no value in the differentiation of Brucella strains in Minnesota. The original oxygen tension requirement,

together with the dye plate growth characteristics, gave fairly reliable means of differentiating the Brucella strains. The Brucella strains isolated in Minnesota from human cases of undulant fever indicated *Brucella suis* to be the etiologic agent about twice as often as *Brucella abortus*.

PULLORUM DISEASE IN TURKEYS. E. P. Johnson and G. W. Anderson. Jour. Inf. Dis., lviii (1936), p. 337.

Two extensive outbreaks of pullorum disease in young poults are reported, the mortality being 100 per cent in one instance and 33 per cent in the other. Indications are that the source of infection was from infected turkey hens. One reactor was found in each of the breeding flocks and *Salmonella pullorum* isolated from the ovary of one bird; both birds reacted in a dilution of 1:400. The authors suggest that this infection is more serious in turkeys than is now regarded by many investigators and that a reaction in a titre above 1:50 is necessary to locate birds harboring *S. pullorum*.

THE BLOOD PH OF LEUKOTIC FOWLS AND THE FILTERABILITY OF THE LEUKOSIS AGENT. E. P. Johnson and W. B. Bell. Jour. Inf. Dis., lviii (1936), p. 342.

The average blood pH of 50 normal fowls was found to be 7.56 and that of 35 leukotic fowls 7.57. Myeloid leukosis and lymphomatosis were transmitted from affected birds to parasite-free birds by injections of bacteria-free filtrates from which the authors conclude there is no foundation to the claims that bacteria and intestinal parasites are etiological factors. Filtration of this agent through membranes demonstrated that it is filtrable through membranes having dimensions of 1,000 to 15,000 millimicrons. A separation of two or more filtrable agents on the basis of particle size as factors in the etiology of the various forms of leukosis was impossible. The author concludes that a single filtrable agent is responsible for the various forms of the disease. The particle size of the leukosis agent lies between the limits of 400 to 100 millimicrons, or possibly less.

73rd Annual Convention A. V. M. A.
Columbus, Ohio, August 11-12-13-14

Regular Army

Lt. Col. Christian W. Greenlee is assigned to Fort Jay, N. Y., for duty effective upon completion of his present tour of foreign service in the Panama Canal Department.

Captain John L. Owens is relieved from further assignment and duty as instructor, Quartermaster Corps Subsistence School, Chicago, Ill., effective on or about July 1, 1936, is then assigned to station at Randolph Field, Tex., and will proceed to Kansas City, Mo., and report to the officer in charge Remount Purchasing and Breeding Headquarters for temporary duty for a period of approximately five months for the purpose of examining remounts for purchase in the Central Remount Purchasing and Breeding Zone; upon completion of this duty to proceed to Randolph Field for duty.

So much of special orders as assigns Major Raymond I. Lovell to duty at Vancouver Barracks, Wash., is amended so as to assign him to duty at the Presidio of Monterey, Calif., upon completion of his present tour of foreign service in the Panama Canal Department.

Captain Arvo T. Thompson is relieved from further assignment and duty at Letterman General Hospital, San Francisco, Calif., is assigned to the Army Medical Center, Washington, D. C., and will sail on the transport scheduled to leave San Francisco on or about August 22, 1936, for New York, N. Y.; upon arrival in New York City will proceed to Washington, D. C., and report to the commanding general, Army Medical Center, for duty.

Major Gerald W. Fitz Gerald is relieved from duty at Presidio of Monterey, Calif., effective in time to proceed to San Francisco, Calif., and sail on the transport scheduled to leave that port on or about November 24, 1936, for the Hawaiian Department. Upon arrival will report to the commanding general for assignment to duty with the Veterinary Corps.

Major Harry J. Juzek is assigned to duty at Vancouver Barracks, Wash., effective upon completion of his present tour of foreign service in the Hawaiian Department.

The appointment of 1st Lieut. Donald Clifford Kelley, Veterinary Corps Reserve, as first lieutenant in the Veterinary Corps, Regular Army, with rank from June 23, 1936, is announced. He will report to the commanding officer, Fort Snelling, Minn., for assignment to duty.

Major Daniel H. Mallan is relieved from his present assignment and duty at Carlisle Barracks, Pa., effective in time for him to proceed to New York, N. Y., and sail on the transport scheduled to leave that port on or about September 16, 1936, for the Panama Canal Department, and upon arrival will report to the commanding general for assignment to duty with the Veterinary Corps.

1st Lieut. Walter T. Carll is directed to proceed to Camp Perry, Ohio, and report on August 2, 1936, to the Executive Officer, National Matches, for temporary duty in connection with the Matches, upon completion to return to his proper station, Brooklyn, N. Y. Lieut. Carll will be camp veterinarian for inspection of meat, meat food and dairy products purchased during the encampment.

Major Seth C. Dildine is relieved from assignment and duty at the New York port of embarkation, Brooklyn, N. Y., effective at such time as will enable him to comply with this order. He will sail on or about January 8, 1937, via Government transportation, from New York, N. Y., to the Philippine Department and upon arrival will report to the commanding general for assignment to duty with the Veterinary Corps in China and will proceed to join that station via Government transportation.

Captain Harvie R. Ellis has been directed to proceed, at the proper time, from Fort Riley, Kan., to Governors Island, N. Y., and report to the commanding general, Second Corps Area, on temporary duty until on or about June 10, 1936, thence via commercial liner, to Europe, on temporary duty in connection with the 1936 Olympic Games at Berlin, Germany, proceeding upon arrival in Europe to Lucerne, Switzerland, on temporary duty for the purpose of training, conditioning and acclimating horses at that place from June 20, 1936 to July 13, 1936, and upon completion proceed to Berlin, Germany, on temporary duty for the purpose of competing in the 1936 Olympic Games, and upon completion return, via commercial liner to New York, N. Y., thence to proper station. Captain Ellis is the official veterinarian for the U. S. Olympic Equestrian Team.

The following-named officers of the Veterinary Corps who have just completed the course of instruction at the Medical Field Service School, Carlisle Barracks, Pa., and are now under orders to proceed to the Army Medical Center, Washington, D. C., for the purpose of pursuing a course of instruction at the Army Veterinary School, are assigned to temporary duty at the stations opposite their names, until such time as will enable them to comply with existing War Department orders.

1st Lieut. Andrew J. Sirilo, West Point, N. Y.
1st Lieut. John H. Rust, Fort Myer, Va.
1st Lieut. Bernard F. Trum, Fort Hoyle, Md.

The following officers of the Veterinary Corps are directed to proceed to Chicago, Ill., and report to the commanding general, Sixth Corps Area, not later than August 4, 1936, for temporary duty during the period of the Second Army Maneuvers, August 8-22, 1936; upon completion of such duty to return to their proper stations.

Major Fred W. Shinn, Fort Francis E. Warren, Wyo.
Major Seth C. Dildine, Surgeon General's Office, Wash., D. C.
Major Harry L. Watson, Fort Sill, Okla.
1st Lieut. Velmer W. McGinnis, Army Medical Center, Washington, D. C.

The above officers are to be assigned to railheads for the purpose of inspection of meat, meat food and dairy products, fresh fruits, and fresh vegetables delivered to these railheads for distribution to the organizations participating in this field Army maneuvers.

Veterinary Reserve Corps

NEW ACCEPTANCES

Cook, Roger Alward.....1st Lt...205 N. Sprague Ave., Bellevue, Pittsburgh, Pa.
Cooperrider, Donald Elmer.1st Lt...2362 Indiana Ave., Columbus, Ohio.
Courter, Robert Davis.....1st Lt...Route 1, Amelia, Va.
Cox, Marion Thomas.....1st Lt...1037 S. Main St., Kenton, Ohio.
Cromley, Curtis William...1st Lt...Route 3, Ashville, Ohio.
Curtin, Charles Joseph.....1st Lt...Livonia, N. Y.
DeLand, Charles William..1st Lt...Temperance, Mich.
Dermody, John Michael....1st Lt...Route 1, Casey, Iowa.
Dodge, Roger Everett.....1st Lt...6 Corning St., Beverly, Mass.

Dougherty, Robert Watson . 1st Lt...Newcomerstown, Ohio.
Dye, Harland Freemont....1st Lt...Bridgewater, N. Y.
Eippert, Paul Emerson.....1st Lt...110 W. Woodruff Ave., Columbus, Ohio.
Elliott, Berton Jasper.....1st Lt...Route 1, Albany, Ohio.
Emminger, Albert Clarence.1st Lt...Route 2, Box 1046, Turlock, Calif.
Frazer, Lloyd Alson.....1st Lt...Route 3, Oskaloosa, Iowa.
Frick, Keith Raymond.....1st Lt...Mio, Mich.
Geyer, Harry George.....1st Lt...Grove City, Ohio.
Glover, Albert Downing....1st Lt. Newark, Mo.
Hackett, Clarence Pierce...1st Lt...Kinsman, Ohio.
Harden, Chester Nahum...1st Lt...11 Lewiston St., Brockton, Mass.
Herman, Leslie Frederick..1st Lt...8016 Whitethorne Ave., Cleveland, Ohio.
Hillstrom, Werner Frederick .....1st Lt...136 Florida St., Laurium, Mich.
Houk, William Sylvanus....1st Lt...402 Park Dr., Muscatine, Iowa.
Hults, Charles Edward.....1st Lt...234 Valley Rd., Ithaca, N. Y.
Jacobs, Marvin Henry.....1st Lt...Cranford Apartments, Ames, Iowa.
Jones, Kenneth Stanton....1st Lt...Rushsylvania, Ohio.
Josse, Charles Knox.....1st Lt...820 Caldwell St., Piqua, Ohio.
Larsen, Burt William.....1st Lt...Cassville, N. Y.
McCutchan, Howard Elbert.1st Lt...Plymouth, Ill.
McNay, Garrett Hillery....1st Lt...Garden Grove, Iowa.
Metz, Clark Allen.....1st Lt...Clarence Center, N. Y.
Mills, John William.....1st Lt...New Paris, Ohio.
Mirin, Solomon.....1st Lt...236 Grand St., Brooklyn, N. Y.
Moore, Morris Edmund....1st Lt...Arcadia, Ind.
Moser, Herman Leroy.....1st Lt...Dalton, Ohio.
Nathanson, Sidney.....1st Lt...115 Dahill Rd., Brooklyn, N. Y.
Nixon, Gaylord Jay.....1st Lt...Route 4, Mansfield, Ohio.
Novy, Vilt James.....1st Lt...Route 3, Box 433, E. St. Louis, Ill.
Pirie, James William.....1st Lt...Springville, Iowa.
Rainey, Paul Alston.....1st Lt...951 Miller Ave., Columbus, Ohio.
Reed, Wesley Raymond....1st Lt...Route 4, Delaware, Ohio.
Richman, Silas.....1st Lt...40 Terrace Ave., Albany, N. Y.
Roscosky, John William....1st Lt...Box 23, Harrison City, Pa.
Sachs, Hyman.....1st Lt...112-10 101st Ave., Richmond Hill, N. Y.
Schmidt, Martin George....1st Lt...Box 6, Pomeroy, Iowa.
Schoentrup, Raymond Vincent .....1st Lt...834 S. Noble St., Shelbyville, Ind.
Schofield, William Carver..1st Lt...741 Binns Blvd., Columbus, Ohio.
Schwiesow, Carleton William .....1st Lt...215 Blackburn St., Ripon, Wis.
Sinai, Harry Joel.....1st Lt...275 Harrison Ave., Jersey City, N. J.
Solt, John Joseph.....1st Lt...Arlington, Ohio.
Spratt, Earl Keith.....1st Lt...306 W. Adams St., Fairfield, Iowa.
Stephan, Sol Galen.....1st Lt...30 Erkenbrecher Ave., Cincinnati, Ohio.
Taylor, Irving Wilson.....1st Lt...DeKalb Junction, N. Y.
Theophilus, Donald Kingston .....1st Lt...Smithland, Iowa.
Thompson, Frederic Bristol.1st Lt...Holland Patent, N. Y.
Twisselmann, Norman Miller .....1st Lt...Santa Maria, Calif.
Venzke, Carl Ebert.....1st Lt...Route 1, Galva, Iowa.
Vogel, William Martin.....1st Lt...15 Evanston Ave., Dayton, Ohio.
Vollmer, Floyd Arthur.....1st Lt...Liberty, Ill.
Wagers, Robert Phillip....1st Lt...Route 1, Scio, Ohio.

Ward, John Arthur.....1st Lt...108 Epsom Course, Hollis, N. Y.
Weaver, Glen John.....1st Lt...Goodrich Rd., Clarence, N. Y.
Weiner, Jules Joseph.....1st Lt...1820 Phelan Pl., New York, N. Y.
Wilson, Wells McCleery....1st Lt...Route 1, Circleville, Ohio.
Zuercher, Vilas Wilford....1st Lt...Dalton, Ohio.
Arron, Daniel Pierce.....1st Lt...312 32nd Ave., Seattle, Wash.
Bills, William Edward.....1st Lt...1597 Hunter St., Columbus, Ohio.
Christopher, Burton Chapman
.....1st Lt...303 E. Union Ave., Olympia, Wash.
Claus, Norman Gustave.....1st Lt...3987 Maybury Rd., Detroit, Mich.
Elsasser, David Sherman...1st Lt...1062 E. 62nd St., Cleveland, Ohio.
Guard, William Paul.....1st Lt...Friday Harbor, Wash.
Henkel, Ernest Lawrence...1st Lt...New Bridge, Ore.
Joneschild, William Raymond
.....1st Lt...616 S. Adams, Tacoma, Wash.
Koutz, Fleetwood Raymond.1st Lt...3224 E. Lombard St., Baltimore, Md.
Legner, Armand Arthur....1st Lt...Leland, Ill.
Williams, George Agusta...1st Lt...Box 61, College Station, Pullman, Wash.
Winiecki, Henry Fred.....1st Lt...702 Cherry St., Raymond, Wash.
Cook, Robert William.....1st Lt...1648 Fairchild St., Manhattan, Kan.
Cope, Russell Parker.....1st Lt...922 Bluemont St., Manhattan, Kan.
Dehner, Loris Arthur.....1st Lt...920 Cedar St., Concordia, Kan.
Gale, Mark Earnest.....1st Lt...332 W. 8th St., Concordia, Kan.
Gouge, Robert Elmer.....1st Lt...400 Dal-Whi-Mo, Sedalia, Mo.
Hinkle, Thomas Clark, Jr...1st Lt...Carbondale, Kan.
Lassen, Keith Obed.....1st Lt...Route 5, Box 514, Phoenix, Ariz.
Lindenstruth, Henry James.1st Lt...305 W. Jackson St., Marshfield, Mo.
Millenbruck, Edgar William 1st Lt...General Delivery, Marysville, Kan.
Murphy, Edward Aloysius..1st Lt...224 S. 11th St., Kansas City, Kan.
Rosenwald, Arnold Samuel.1st Lt...870 Crenshaw Blvd., Los Angeles, Calif.
Tellejohn, Arthur Louis....1st Lt...1601 Garfield Ave., Kansas City, Kan.
Twiehaus, Marvin John, Jr..1st Lt...Route 4, Box 675, Independence, Mo.
West, John Leslie.....1st Lt...1208 Kearney St., Manhattan, Kan.
Bundalian, Martin Fran-
cisco .....1st Lt...222 Sylvia, Ermita, Manila, P. I.
Crawford, James Pingree..1st Lt...722 State Office Bldg., Lansing, Mich.
Jensen, Christian Godtfred.1st Lt...c/o T. S. Rich, 722 State Office Bldg., Lansing, Mich.
Anderson, Wayne Allen....1st Lt...704 S. College Ave., Fort Collins, Colo.
Broadwell, John Marshall..1st Lt...Box 645, Glenwood Springs, Colo.
Cardona, Carlos Juan.....1st Lt...2736 Purington Ave., Fort Worth, Tex.
Coleman, Charles Leonard..1st Lt...200-B E. Chestnut St., Glendale, Calif.
Coop, Moray Claude.....1st Lt...1625 Broadway, San Antonio, Tex.
Cowen, Russell Lee.....1st Lt...Rainbow Blvd., Salida, Colo.
Cox, Charles Edward.....1st Lt...Route 1, Auburn, Ala.
Dixon, Albert Vernon.....1st Lt...Route 1, Quitman, Ga.
Exley, Eldred Walton.....1st Lt...Route 1, Clyo, Ga.
Hester, Kenneth Jack.....1st Lt...Eaton, Colo.
Jones, Leslie Wakelin.....1st Lt...Route 1, Box 179, Littleton, Colo.
Kinchelow, William Davis..1st Lt...2426 Lafayette St., Denver, Colo.

Landers, Leo.....1st Lt...4 Wellington Hill St., Boston, Mass.
Leonard, Harry Alfred.....1st Lt...Holly, Colo.
Maxwell, Ralph Arnott.....1st Lt...Salem, N. Y.
Milici, Rosario Francis....1st Lt...1041 60th St., Brooklyn, N. Y.
Mood, Bernard Farr.....1st Lt...Route 4, Box 59, Savannah, Ga.
Myers, Philip.....1st Lt...Floresville, Tex.
Nomura, Paul Talogi.....1st Lt...Box 758, Honolulu, T. H.
Quortrup, Erling Richard..1st Lt...College Station, Tex.
Smith, Hugh Dowd.....1st Lt...Conover, N. C.
Smith, John William.....1st Lt...Stockyards Station, Route 1, Denver, Colo.
Utley, Thomas Edmund....1st Lt...Dawson, N. M.
Wilbanks, Carlos Stanford..1st Lt...Clarkesville, Ga.
Zedlitz, Alfred Charles.....1st Lt...1203 8th St., Ballinger, Tex.
Alkire, Robert Louis.....1st Lt...Box 277, Webster City, Iowa.
Baum, Harvey.....1st Lt...Route 2, East Sparta, Ohio.
Benton, Carl Ralph.....1st Lt...139 Vernal St., Everett, Mass.
Berliner, Meyer.....1st Lt...6 Margot Pl., Great Neck, L. I., N. Y.
Bradley, Donald Herbert...1st Lt...1569 N. High St., Columbus, Ohio.
Brown, Robert Frederick...1st Lt...Bangor, N. Y.
Brown, Robert James.....1st Lt...Akron, Iowa.
Burch, George Edward.....1st Lt...Granville, N. Y.
Cairy, Clyde Frank.....1st Lt...4900 Morningside Ave., Sioux City, Iowa.
Cole, Elvin George.....1st Lt...Kamas, Utah.
Collins, Floyd Merle.....1st Lt...Route 2, Tilden, Neb.

PROMOTIONS

Apple, Henry Jackson.....1st Lt...1037 Chambers Rd., Columbus, Ohio.
Brown, Roswell Leo.....1st Lt...1817 Church St., Evanston, Ill.
Gingras, George Edward...1st Lt...Fort Covington Rd., Malone, N. Y.
Green, Charles Frederick, Jr. .....1st Lt...E. Genesee St., DeWitt, N. Y.
Guthrie, Richard Sidney....1st Lt...Box 18, Hillsdale, N. Y.
Krill, Walter Roland.....1st Lt...Veterinary Clinic, O. S. U., Columbus, Ohio.
McFadden, Glenn Melvin...1st Lt...Natoma, Kan.
Micuda, John.....1st Lt...General Delivery, Laurel, Md.
Thiele, Mervin Ivan.....1st Lt...Prescott, Iowa.
Thomas, Owen Ellis.....1st Lt...Box 54, 203 Granville Rd., Granville, Ohio.

NEW ASSIGNMENTS TO ACTIVE DUTY WITH CCC

Morgan, Donald R.....1st Lt...Vancouver Barracks, Wash.
Nieberding, Joseph F.....1st Lt...Fort Worth Dist., Fort Worth, Tex.

TERMINATION OF ASSIGNMENT TO ACTIVE DUTY

Martin, Earl T.....Capt...Redding, Calif.
Buell, Herbert J.....1st Lt...Fort Totten, N. Y.
Kelley, Donald C.....1st Lt...Fort Snelling, Minn. (Appointed 1st Lieut. in Regular Army).

73rd Annual Convention A. V. M. A.
Columbus, Ohio, August 11-12-13-14

Woman's Auxiliary to the Virginia State Veterinary Medical Association

The annual meeting of the Woman's Auxiliary to the Virginia State Veterinary Medical Association was held at the Virginia Polytechnic Institute, Blacksburg, July 9-10, with 32 members and 28 visitors in attendance.

After a delightful luncheon, at the Faculty Center, followed by an enjoyable musical program, the meeting was called to order by the president, Mrs. P. M. Graves, Culpeper. The minutes of the previous meeting were read and approved. Following the roll-call and collection of dues, the committee chairmen read their reports, which were approved and ordered placed on file.

The Auxiliary, for several years, has given $25.00 to the Sheltering Arms Hospital, Richmond, a state-wide charitable institution. After some discussion, a committee was appointed to decide on the disposition of the money this year, should a more worthy cause be found.

An inspiring paper was read by Mrs. R. T. Gregory, Fredericksburg, on "How the Veterinarian's Wife Can Be of Help to Him." This type of discussion in the meeting was an innovation and was thoroughly enjoyed by all.

The following officers were elected for the ensuing year: President, Mrs. P. M. Graves, Culpeper; first vice-president, Mrs. E. H. Drake, Leesburg; secretary, Mrs. Taylor P. Rowe, Richmond; treasurer, Mrs. P. F. Galloway, Richmond; assistant secretary, Mrs. H. C. Givens, Richmond; assistant treasurer, Mrs. I. P. Gilbert, Courtland; historian, Mrs. H. H. Rowe, Richmond, and Auxiliary advisor, Mrs. R. E. Brookbank, Richmond.

After the meeting was adjourned, the ladies were conducted on a tour of the campus, where many impressive buildings are to be seen. The tour terminated at the garden of Mrs. I. D. Wilson, who gave a delightfully refreshing tea in honor of the Woman's Auxiliary. The day ended pleasantly with the annual banquet, where the speeches were interesting but brief, a welcome factor on an extremely warm night.

On Friday, July 10, everyone was up by 9 o'clock and after breakfast, cabs were supplied for the drive to beautiful Mountain Lake, a well-known resort of Virginia. Upon returning to Blacksburg from Mountain Lake, the ladies parted to prepare for the return journey home. Each one agreed that she had had a grand time and promised to meet again next year.

ANN ELLIOTT ROWE, *Secretary.*

Bicentennial Campaign at Pennsylvania

Plans for a Bicentennial Campaign in which the University of Pennsylvania will seek the sum of $6,400,000 to meet the general needs of the University, and an additional sum of $6,100,000 for the support of special projects, have been approved by the Trustees, as announced recently by President Thomas S. Gates. The $12,500,000 campaign will begin in the fall of this year and will be brought to a close before the University celebrates its 200th anniversary in 1940.

As numerated by President Gates, the most pressing general needs of the University and the specific sums which will be sought to meet them are:

(1) Endowment for improvement of teaching in the undergraduate schools and in the Graduate School, $3,600,000.
(2) Endowment for improvement of teaching and research in the professional schools, $800,000.
(3) General University endowment, $2,000,000.

According to President Gates, "Development of teaching and research work in specialized fields of medicine and dentistry, and of research work in animal pathology, constitute further important objectives for which the University will seek funds in the interest of its professional schools of Medicine, Graduate Medicine, Dentistry and Veterinary Medicine."

Pennsylvania Membership in the A. V. M. A.

Year	*Members*	*Year*	*Members*
1923	178	1930	337
1924	178	1931	334
1925	184	1932	303
1926	186	1933	283
1927	221	1934	260
1928	242	1935	247
1929	327	1936 (July 31)	261

STATE VETERINARY MEDICAL ASSOCIATION OF TEXAS

The thirteenth semi-annual meeting of the State Veterinary Medical Association of Texas was held at Texas A. & M. College, College Station, June 2-4, 1936, with President Chas. W. Neal, of San Antonio, presiding.

More than 60 members of the Association and 14 members of the Ladies Auxiliary were officially welcomed to the college by Dr. Mark Francis, dean of the School of Veterinary Medicine and Surgery. Other speakers during the morning included Drs. Chas. W. Neil; Frank Hecker, Houston; I. B. Boughton and W. T. Hardy, Sonora, and T. O. Booth, H. L. Darby, and A. K. Kuttler, Fort Worth.

During a short business session, Dr. M. E. Gleason, of San Antonio, was elected as alternate to the A. V. M. A. House of Representatives. He will act only in the absence of Dr. J. K. Northway, Kingsville, delegate from Texas. An invitation from Fort Worth to hold the annual meeting there in January was accepted. Houston and Brownwood also extended invitations. A subject of much interest was the question of out-of-state veterinarians practicing at Texas race-tracks without a Texas license. After much discussion, the question was referred to the State Board of Veterinary Examiners, who will seek a ruling on the matter from the Attorney General.

The Wednesday afternoon clinical session was conducted by Drs. M. E. Maier, Orange; A. E. Wharton, R. C. Dunn and A. A. Lenert, College Station; Chas. W. Neal and Mr. John Miliff, senior veterinary student.

Thursday, Dr. R. A. Self, of Dallas, conducted a clinic on small animals in the morning, and Dr. A. A. Lenert conducted a clinic on horses and mules in the afternoon. Among those participating in the clinical work were four A. & M. graduates who completed their state board examinations for licenses on June 2. The four were Drs. C. M. Coop, B. F. Mood, G. T. Eads and C. L. Coleman. Drs. Coop and Mood performed a cecectomy. Others

taking part in the clinic included Drs. W. R. McCuistion, Fort Worth; G. E. McIntosh, Arlington; R. C. Dunn and R. P. Marsteller, College Station; H. W. Laughlin, New Iberia, La., and R. A. Self.

The Ladies Auxiliary opened its meeting Tuesday with an executive session in the afternoon and a pioneer dinner in the evening, honoring Mrs. Mark Francis. Wednesday, the Auxiliary members were guests at a beautifully appointed luncheon tendered them by the ladies of the faculty at the picturesque old town of Navasota. They viewed the beautiful statue of La Salle and other points of interest, then motored on to old Washington on the Brazos, spending the afternoon there.

The Association and the Ladies Auxiliary entertained in the evening, beginning at 7:30 o'clock, with a barbecue and dance in the Veterinary Hospital.

D. PEARCE, *Secretary.*

MICHIGAN STATE VETERINARY MEDICAL ASSOCIATION

The fifty-fourth annual meeting of the Michigan State Veterinary Medical Association was held at Michigan State College, East Lansing, June 23-24, 1936.

During the forenoon of the first day, the time was devoted to an out-door clinic. This was conducted by Drs. J. P. Hutton, Michigan State College; T. A. Sigler, Greencastle, Ind., and C. E. Hagyard, Lexington, Ky. They were assisted by Drs. E. K. Sales, East Lansing; A. E. Erickson, Charlotte; F. E. Stiles, Battle Creek, and E. C. W. Schubel, Blissfield.

Dr. Stiles and Dr. Schubel supplied two cases for the clinic. Dr. Stiles' case was an aged horse which was a roarer, and Dr. Schubel's case was a colt, less than a year old, also a roarer. Dr. Sigler operated on both cases.

Tuesday afternoon, Dr. Sigler discussed breeding problems of cattle. Dr. Hagyard discussed breeding problems in horses as he has encountered them in his practice in Kentucky. J. J. Arnold, a senior veterinary student, demonstrated the pregnancy test for mares, and Dr. H. J. Stafseth, of Michigan State College, discussed streptococcic infections in dogs.

A banquet for the veterinarians and their wives was held in the evening. The entertainment was provided by Dr. B. J. Killham, of Michigan State College, who had a very novel program arranged for the entertainment of the visitors.

On Wednesday, Dr. Sigler discussed digestive diseases of cattle. Dr. Hagyard discussed parasitism in horses, stressing the danger of strongyles, especially in colts. Prof. George A. Brown, of Michigan State College, and Dr. F. E. Stiles discussed sheep feeding and diseases of sheep.

Dr. W. P. S. Hall, Superintendent of the Bureau of Food and Sanitation, Division of Health, Toledo, Ohio, discussed meat inspection, emphasizing the fact that all meat inspection positions should be held by veterinarians who are trained for such work. Mr. L. M. Board, sanitary engineer, of Hillsdale, spoke on public health education.

Much of the afternoon was spent in a lively discussion of affairs of the Association and the election of officers. The following officers were elected for the ensuing year: President, Dr. J. Wm. G. Hansen, Greenville; first vice-president, Dr. A. Z. Nichols, Hillsdale; second vice-president, Dr. D. A. Curtis, Breckenridge; third vice-president, Dr. F. M. McConnell, Litchfield; secretary-treasurer, Dr. E. C. W. Schubel, Blissfield.

Dr. Arthur McKercher, Lansing, was elected to the Board of Directors for six years to succeed himself, and Dr. A. T. McIntyre, Brown City, was elected to the Board for a term of five years, to succeed Dr. Schubel.

Dr. J. Wm. G. Hansen, of Greenville, was elected as delegate to the A. V. M. A. House of Representatives and Dr. Ward Giltner, of East Lansing, was elected as alternate.

E. C. W. SCHUBEL, *Secretary.*

GEORGIA STATE VETERINARY ASSOCIATION

The thirtieth annual meeting of the Georgia State Veterinary Association was held at the Oglethorpe Hotel, Brunswick, July 2-3, 1936, with Dr. Charles C. Rife, of Atlanta, presiding.

Mr. R. C. Job, secretary of the Brunswick Board of Trade, welcomed the visitors to Brunswick and gave a brief résumé of the history of Brunswick, which was celebrating its bicentennial. Dr. E. D. King, of Valdosta, very ably responded to the welcoming address.

In his presidential address, Dr. Rife stressed the desirability of state associations coöperating in the matter of fixing their meeting dates so that associations in adjacent states could secure men of national reputation for their programs, with all of the advantages that would result from such an arrangement.

Dr. J. C. Flynn, president of the American Veterinary Medical Association, spoke on the subject, "The Veterinary Profession,"

and incidentally told of some of the activities of the national organization. Dr. I. S. McAdory, of the Alabama Polytechnic Institute, also contributed to the morning program. Hon. Columbus Roberts, of Columbus, was present with his son-in-law, Dr. Jesse Miller, and spoke on the live stock industry of Georgia and the necessity of maintaining good pastures and grasses.

At the afternoon session, Dr. I. S. McAdory read a paper on "Clinical Operations for Paralysis of the Vocal Cords." Dr. McAdory dwelt on the complications which sometimes follow these operations and many of the practitioners entered into the lively discussion which followed, telling of their experiences with this condition. A round-table discussion followed on the following subjects: Heart-worm disease, screw-worm infestation, fractures in small animals, and acetonemia in cattle.

The banquet was held at 6:30, with Dr. Peter F. Bahnsen acting as toastmaster. The after-dinner speakers were Drs. Flynn and McAdory, and Hon. Columbus Roberts, all of whom made their addresses very short so that the veterinarians and their families could attend the historical pageant marking the bicentennial of the founding of Brunswick, which was held at Saint Simons Island at 8:30.

The morning of the second day was turned over to Dr. Flynn who conducted a clinic. He demonstrated methods of restraint, sutureless spaying, and anesthesia. Several cases were presented for diagnosis and treatment. The clinic was well attended and proved helpful to everybody present. Dr. Flynn concluded the clinic by discussing canine distemper at length, including the various methods of immunization.

At the business session which concluded the meeting, the following officers were elected for the ensuing year: President, Dr. R. D. Carr, Thomasville; vice-president, Dr. R. O. Barnes, Claxton, and secretary-treasurer, Dr. J. E. Severin, Atlanta. Columbus was chosen as the meeting place for 1937.

C. C. RIFE, *President.*

NORTH CAROLINA STATE VETERINARY MEDICAL ASSOCIATION

Approximately 50 North Carolina veterinarians attended the thirty-fifth annual meeting of the North Carolina State Veterinary Medical Association, which convened at Goldsboro, July 7-8, 1936.

Dr. W. A. Carter, of Weldon, president of the Association, introduced Dr. T. A. Sigler, of Greencastle, Ind., past president of the American Veterinary Medical Association, who gave a general discussion of "Equine Practice." He recommended for summer sores, both as a preventive and a curative, glycerin 85 parts, oil of tar 10 parts, and phenol 5 parts. At the large-animal clinic, held on the second afternoon, Dr. Sigler performed the roaring operation on mules, did a tenotomy, and enucleated a tumefied eyeball, all under chloral hydrate anesthesia, given intravenously.

Dr. H. B. Smith, of Farmville, read a paper on "Old Theories and New Practices in Veterinary Medicine." He advocates sodium chloride, soda, and potassium permanganate as a treatment for wounds. "Rabies" was discussed from the laboratory standpoint by Dr. John H. Hamilton, Director, State Laboratory of Hygiene, Raleigh. He presented charts showing that in North Carolina the prevalence of rabies runs in ascending and descending curves, with the peaks about six or seven years apart. From his observation, the disease occurs uniformly throughout the year. He stated that North Carolina had not made any progress in the control of rabies during the last 25 years. Dr. Hamilton said that veterinarians know a case of rabies when they see it; that he would rely on this clinical observation even when the laboratory examination is negative.

Dr. J. C. Flynn, Kansas City, Mo., president of the American Veterinary Medical Association, conducted a canine clinic the first afternoon and demonstrated the sutureless spaying operation, and the Thomas appliance for fractures. On the morning of the second day, Dr. Flynn discussed the care and management of canine matrons and pups. He recommended the following prescription for caked mammary glands and also to dry up the secretion of milk: tincture of belladonna 2 ounces, spirits of camphor 2 ounces, tincture of opium 1 ounce. Dr. M. M. Leonard, Asheville, discussed his method of handling mastitis.

At the banquet held at Hotel Goldsboro, Dr. W. T. Scarborough, Raleigh, served as toastmaster and presented Dr. J. N. Johnson, Goldsboro, member of the State Board of Health, who gave an address on the relationship existing between the medical, dental and veterinary professions and suggested that they ought to meet together occasionally.

Dr. A. A. Husman, Raleigh, delegate to the A. V. M. A. House of Representatives, reported at the business meeting what took place in the House of Representatives meeting at Oklahoma City last year.

An amendment to the by-laws was introduced to permit a veterinarian, who is in good standing in a state veterinary association, to become a member of the North Carolina Association without the payment of an entrance fee.

Officers elected for the ensuing year were: President, Dr. B. J. Lindley, Winston-Salem; first vice-president, Dr. G. A. Ferguson, Reidsville; second vice-president, Dr. B. M. Weston, Asheboro; secretary-treasurer, Dr. J. H. Brown, Tarboro (reëlected), and directors, Dr. M. M. Leonard, Asheville, and Dr. T. A. Monk, Goldsboro.

Wrightsville Beach was selected for the 1937 annual meeting.

J. H. BROWN, *Secretary.*

VIRGINIA STATE VETERINARY MEDICAL ASSOCIATION

The forty-third annual meeting of the Virginia State Veterinary Medical Association was held at Virginia Polytechnic Institute, Blacksburg, July 9-10, 1936. The meeting was called to order by Dr. I. D. Wilson, President, in Agricultural Hall.

The attendance was excellent, 160 veterinarians, their wives and friends being registered. Visitors were in attendance from Maryland, the District of Columbia, West Virginia and Tennessee.

Great interest was manifested in the papers presented so ably by those whom we were fortunate in having on our program. Dr. John D. Beck, University of Pennsylvania, Philadelphia, Pa., had as his subject, "Rabies Challenge." Dr. J. H. Rietz, West Virginia University, Morgantown, W. Va., discussed "Parasites and Their Control."

Dr. W. T. Miller, U. S. Bureau of Animal Industry, National Research Center, Beltsville, Md., presented "Observations in Bovine Mastitis." This being a subject of vital importance to every veterinarian, an animated discussion followed. Dr. E. C. McCulloch, Pennsylvania Salt Manufacturing Company, Philadelphia, Pa., spoke on "Disinfectants and Their Uses," which was very interesting and instructive.

Dr. A. E. Wight, Chief, Tuberculosis and Bang's Disease Eradication Division, U. S. Bureau of Animal Industry, Washington, D. C., discussed briefly the progress being made in Bang's disease eradication in the United States.

A business meeting followed with important matters being brought up for discussion and settlement. One important point

was an amendment to the by-laws, unanimously approved and adopted, which was as follows:

Chapter IV, Fees and Dues:

Article 1—Add: "Veterinarians regularly employed by the United States Government, who are members of the American Veterinary Medical Association, may be elected to membership without payment of initiation fee."

The election of officers for the ensuing year resulted as follows: President, Dr. I. P. Gilbert, Courtland; first vice-president, Dr. L. K. Spitler, Luray; second vice-president, Dr. O. F. Foley, Harrisonburg; secretary, Dr. A. J. Sipos, Richmond (reëlected), and treasurer, Dr. R. E. Brookbank, Richmond (reëlected).

Following the business meeting, we proceeded to the banquet hall, where a most pleasant two-hour period was spent.

In the absence of Dr. J. C. Flynn, president of the American Veterinary Medical Association, who was scheduled for the small-animal clinic and demonstration, but who suddenly became ill during the night of July 9, Dr. Taylor P. Rowe, of Richmond, with other local talent, very ably "pinch hit" for Dr. Flynn.

Thirteen applicants took the State Board examination on July 9.

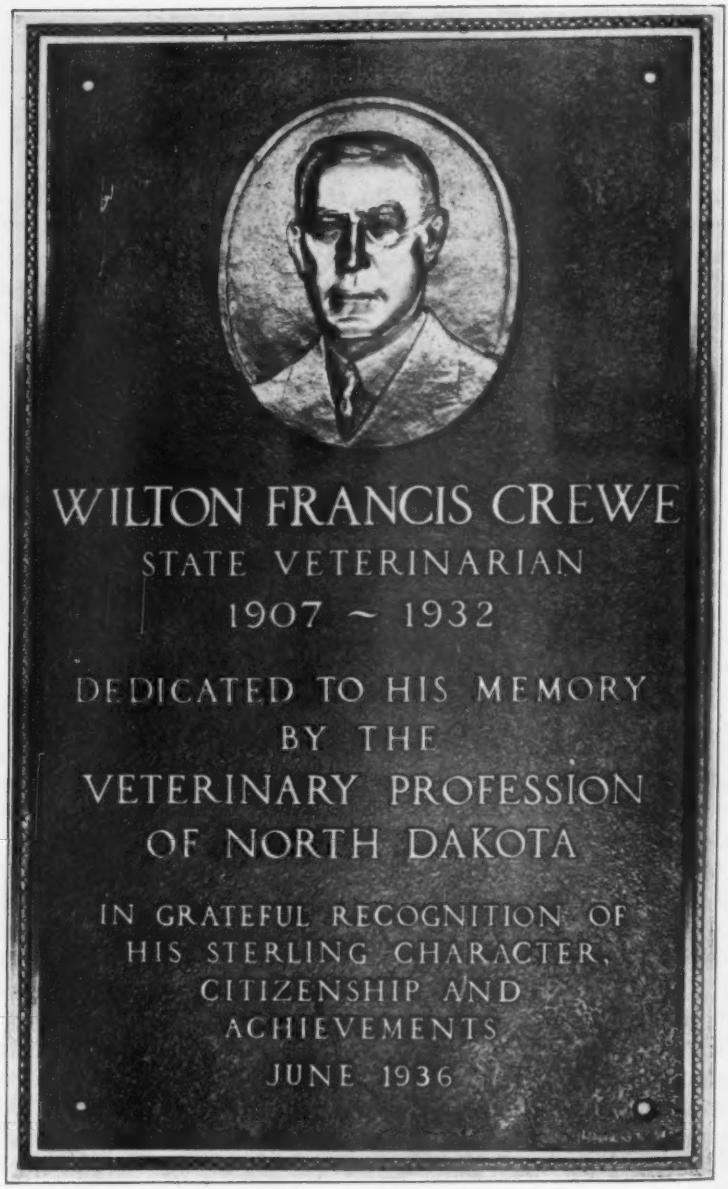
A. J. SIPOS, *Secretary.*

NORTH DAKOTA VETERINARY ASSOCIATION

The thirty-second annual meeting of the North Dakota Veterinary Association was held at the Agricultural College, Fargo, July 14-15, 1936, with the President, Dr. R. R. Cusack, Jamestown, in charge.

A program of interesting and instructive papers and discussions was presented which included the following subjects: "Municipal Milk Inspection," by Dr. H. P. Roberts, Fargo Health Department; "Swine Erysipelas" and "Observations on the Prevention of Certain Poultry Diseases," by Dr. L. Van Es, University of Nebraska, and "Pregnancy Disease of Sheep," by Dr. Lee M. Roderick, Fargo. The subject of "Pullorum Disease" was discussed by Dr. J. V. Miles, Ellendale, with a demonstration of his technic. An informal discussion was held on experiences with equine encephalomyelitis during the 1935 epizoötic. The clinical demonstrations were given by Drs. L. A. Benson, Grand Forks; R. E. Shigley, Minot, and T. O. Brandenburg, Bismarck.

The feature of the meeting was the evening banquet session, held at the Gardner Hotel, with an attendance of 78 veterinar-



TABLET IN MEMORY OF DR. WILTON FRANCIS CREWE

ians, their wives and invited guests. Dr. J. W. Robinson, Garrison, presided in an especially pleasing manner. Governor Walter Welford was presented and responded with an enthusiastic address which was particularly appropriate for the occasion. Dr. L. Van Es, in a fitting address on the subject, "Live Stock Sanitation as a Function of Government," then presented a bronze memorial tablet from the Association to the North Dakota Live Stock Sanitary Board, to commemorate the life and work of the late Dr. Wilton Francis Crewe, who served efficiently as State Veterinarian for over 25 years. Mr. W. L. Richards, Killdeer, president of the Live Stock Sanitary Board, accepted the tablet for the Board in a fitting way.

In spite of the intense heat and the desperate drouth which prevailed, the meeting was a success. The following officers were elected for the ensuing year: President, Dr. A. M. Brolling, Fargo; vice-president, Dr. Walter Fleenor, Fairmount, and secretary-treasurer, Dr. Lee M. Roderick, Fargo.

LEE M. RODERICK, *Secretary.*

MAINE VETERINARY MEDICAL ASSOCIATION

The summer meeting of the Maine Veterinary Medical Association was held at Sanford, July 16, 1936. This meeting was one of the first to be held in the form of a clinic and was attended by 20 Maine veterinarians, seven from New Hampshire, and several visitors. A very interesting program was arranged by Dr. C. E. Dutton, of Sanford, and included operations and demonstrations on both large and small animals during the afternoon.

The large-animal clinic was held at Mr. Harold Shaw's farm and Mr. Charles Trafton's horse sales stables, and the small-animal demonstrations were conducted at Dr. Dutton's hospital. The members took much interest in the demonstrations and aided greatly by giving help on the program.

The ladies spent the afternoon visiting various beaches and places of interest along the coast in a bus chartered by the Association for the occasion. At 7 p. m., a shore dinner was held at Dunstan, and was attended by 20 veterinarians and their wives.

At the business meeting, Dr. George M. Potter, of Portland, was elected delegate to the A. V. M. A. House of Representatives and Dr. Paul R. Baird, of Waterville, was elected alternate.

It is hoped that in the future the Association will maintain this type of meeting annually, as the members are very much in favor of it.

S. W. STILES, *Secretary.*

NECROLOGY

MARK FRANCIS

Few veterinarians in this country have received the recognition that was accorded Dr. Mark Francis, of College Station, Texas, who died June 28, 1936. On numerous occasions during recent years, he had been honored and fêted, in recognition of the service he had rendered the live stock industry of the South in general and his adopted state in particular.

On the occasion of the 75th anniversary of the founding of Ohio State University, President W. O. Thompson paid Dr. Francis the following tribute:

> If Ohio State University had trained but one man in the 75 years of its existence, and that man was Dr. Mark Francis, of Texas, it had given back to its people more than they had expended upon it in the three-quarters of a century of its existence.

Last year, Dr. Francis was tendered a birthday party by the Texas A. & M. College Student Chapter of the A. V. M. A. On that occasion, Dr. T. O. Waltham, president of Texas A. & M. College, praised Dean Francis as a scientist, a gentleman, a philosopher and a man. He said:

> The man to whom you do honor this evening deserves every honor that you can bestow upon him. It seems to me that his life is one that has been lived in accord with a plan and a harmoniousness of purpose definitely directed toward the achievement of a great ideal; one in which the objective has never been lost sight of; one where selfishness has been subverted to service; one in which the major interests of a great mind and character have been dedicated to the service of his fellow man.

The same year, Dr. Francis was honorary vice-president of the Southwest Exposition and Fat Stock Show, held at Fort Worth, Texas, and honor guest at the annual dinner of the Texas Hereford Breeders Association. On that occasion, he was presented with a scroll by that organization in coöperation with the Texas and Southwestern Cattle Breeders Association. The scroll read:

> In recognition and sincere appreciation of the service he has rendered the live stock industry of Texas and the entire country, his many years of research on Texas fever and his successful work in overcoming this dreaded disease and making it possible to improve our cattle with better blood, the value of his scientific

research regarding other animal diseases and the important economic bearing it had on the live stock industry, his great work as a teacher and scientist in the field of veterinary medicine and surgery, his unselfishness, his devotion to duty, his common sense philosophy, and his admirable qualities as a man and citizen whose influence for good has been so pronounced among the

DR. MARK FRANCIS

thousands of students and live stock men to whom he has lectured during the half-century he has served the A. & M. College of Texas, this scroll is respectfully dedicated with highest esteem and affection to Dr. Mark Francis, D.V.M., LL.D., Dean of the School of Veterinary Medicine and Science of the Agricultural and Mechanical College of Texas, College Station, Texas.

Born at Shandon, Ohio, March 19, 1863, Dr. Francis was graduated from the New London, Ohio, High School and then entered

Ohio State University for the study of veterinary medicine. He was graduated in 1887, the only member of his class, and the first to receive a veterinary degree from the institution. The following year, he attended the American Veterinary College, in New York City, taking postgraduate work in anatomy. He also studied at the University of Michigan and in Hannover, Germany. In 1929, he was awarded the LL.D. degree by the University of Miami, of Oxford, Ohio.

Dr. Francis joined the staff of Texas A. & M. College in 1888, as professor of veterinary medicine in the School of Agriculture. In 1916, when the School of Veterinary Medicine was organized as a separate division of the College, Dr. Francis was made dean of the school.

Shortly after his arrival in Texas, Dr. Francis interested himself in the disease then generally known as Texas fever. Dr. Francis attacked the problem on two fronts. First, experiments were conducted with a view to immunizing northern cattle against the disease, so that they could be shipped to the South with reasonable safety, for the purpose of improving the quality of southern cattle. The first experiments consisted of the injection of serum obtained from immune southern cattle, on the theory that the blood contained something in the nature of an antitoxin. These experiments failed to produce any appreciable immunity in the treated animals. Next, attempts were made to produce immunity in young cattle by injecting them with virulent blood, this being done during the fall or winter, when an attack of Texas fever would not necessarily be fatal. This experiment was encouraging from the start. About 20,000 head of cattle were thus treated under the direction of Dr. Francis and the death rate, which previously ran as high as 50 or even 80 per cent, was reduced to approximately 5 or 6 per cent. Subsequent experiments were designed with a view to destroying the tick that had been shown to play an important part in the spread of the disease.

Dr. Francis joined the A. V. M. A. in 1897. He served as resident secretary for Texas, 1897-03, 1906-07 and 1910-12; as a member of the Committee on Intelligence and Education, 1907-08, and as a member of the Committee on the Revision of Anatomical Nomenclature, 1918-23. He was a member of the State Veterinary Medical Association of Texas and served as president of that organization for two years (1902-04). The 1934 semi-annual meeting was designated the Mark Francis Meeting, in honor of Dr. Francis. He was a member of the Twelfth International Veterinary Congress and the American Association for the Advance-

ment of Science. For several years, he had been a life member of the Texas Academy of Science.

Dr. Francis was known nationally through his work in collecting fossils of animal life that flourished in prehistoric days. This collection is one of the largest in the country, and for years has been an attraction for visitors to the college, where it is housed in Francis Hall, named in his honor.

ROBERT CLYDE MYLNE

Dr. Robert C. Mylne, of McMinnville, Ore., died suddenly at his home on May 30, 1936. He had been in poor health for some time, but had improved slightly, so that his death was unexpected.

Born in Smiths Falls, Ontario, November 10, 1857, Dr. Mylne attended the Ontario Agricultural College and was graduated in 1880. Later he decided to study veterinary medicine and entered the Montreal Veterinary College. He completed the course at that institution in 1889, and one year later received his D.V.S. from McGill University. Dr. Mylne practiced first in Indiana. In 1900 he removed to Polo, Ill., and in 1911 to McMinnville, Ore.

Dr. Mylne joined the A.V.M.A. in 1929. He was a member of the Oregon State Veterinary Medical Association and the Willamette Valley Veterinary Medical Association. He is survived by his widow (née Jessie L. Schryver), two sisters, a brother, two daughters and one son.

JUSTIS R. KUHNS

Dr. Justis R. Kuhns, of Wilmington, Del., died at his home, June 26, 1936, after an illness of nearly 19 months, caused by injuries received in an automobile accident in November, 1934.

Born near Quakertown, Pa., October 6, 1868, Dr. Kuhns attended one session at the Ontario Veterinary College, 1895-96, but was never graduated from that institution. In 1906, he took a short course at the Chicago Veterinary College, but was never able to qualify as a graduate before the Delaware State Board of Veterinary Examiners. He practiced first in Dover, Del., but removed to Wilmington in 1914. He was a member of the Odd Fellows, the Red Men's Lodge of Dover, the Elks and Kiwanis Club.

C. J. HENDERSON

Dr. C. J. Henderson, of Newman Grove, Neb., died in a hospital at Norfolk, Neb., June 30, 1936. He had been taken there for treatment and had been seriously sick less than a week, although he had been a sufferer from diabetes for several years. A streptococcic infection is believed to have been the immediate cause of death.

Born at Lake Mills, Iowa, July 10, 1892, Dr. Henderson was a graduate of the Saint Joseph Veterinary College, class of 1919. He was also a graduate in pharmacy. He had practiced at Newman Grove ever since his graduation, having purchased the practice of Dr. L. Q. Bulla (K. C. V. C. '11), in 1919.

Dr. Henderson joined the A. V. M. A. in 1922.

WILLIAM C. HANAWALT

Dr. William C. Hanawalt, of Hamilton, Ill., died at his home, June 28, 1936, after an extended illness.

Born in Iowa, June 19, 1859, Dr. Hanawalt was a graduate of the Chicago Veterinary College, class of 1892. He practiced at several places in Illinois, including Sheffield, Galesburg, Quincy, Hull, Payson and Hamilton, and in Hannibal, Mo.

Dr. Hanawalt was a member of the Modern Woodmen and the Odd Fellows. He is survived by his widow (née Alpha Barton), two sons, one sister and two brothers.

OSCAR ALLEN KYLE

Dr. O. A. Kyle, of Bloomington, Ill., died at Saint Joseph's Hospital, that city, July 14, 1936, as a result of heat prostration, according to a coroner's verdict. He had been in an automobile accident a few hours before his death.

Born at Highland, Ill., November 1, 1874, Dr. Kyle was graduated from high school and the Chicago Veterinary College (1901). He immediately located in Bloomington and practiced there until his death.

Dr. Kyle joined the A. V. M. A. in 1919. He was a member of the Illinois State Veterinary Medical Association and the McLean County (Ill.) Veterinary Association. He is survived by two sisters and four brothers, three of whom are veterinarians: Dr. Albert Kyle (Chi. '92), of Highland, Ill.; Dr. Melvin

Kyle (Chi. '96), of Chatsworth, Ill., and Dr. N. W. Kyle (Chi. '96), of Colfax, Ill.

CHARLES THOMAS FREY

Dr. Charles T. Frey, of Hills Grove, R. I., died at his home, July 3, 1936, after suffering from heart trouble for five years.

Born in Erie, Pa., April 2, 1864, Dr. Frey was graduated from the Ontario Veterinary College in 1899. He located at West Warwick, R. I., and practiced there until five years ago, when he was obliged to retire on account of ill health.

Dr. Frey joined the A. V. M. A. in 1910. He was a member of the Rhode Island Board of Registration in Veterinary Medicine for 20 years and was chairman at the time of his death. He was an Odd Fellow and a member of Central Grange No. 34. He is survived by his widow (née Jane Gaddis), two brothers and one sister.

J. G. F.

FRANK THOMAS OWENS

Dr. Frank T. Owens, of Pontiac, Ill., died at his home, July 10, 1936, following an illness of six weeks.

Born at Chalmers, Ind., February 21, 1894, Dr. Owens was educated in the schools of Delphi, Ind. He was a graduate of the Indiana Veterinary College, class of 1923, and practiced in Indianapolis until about two years ago, when he removed to Pontiac, Ill.

Dr. Owens was a member of the Illinois State Veterinary Medical Association and the Knights of Columbus. He is survived by his widow (née Grace Pigman), one son, his father, two sisters and three brothers, one of whom, Dr. J. A. Owens, of El Paso, Ill., is a veterinarian.

JAMES H. McLEAN

Dr. J. H. McLean, of La Porte, Ind., died at his home, July 13, 1936. He had been in failing health for some time.

Born at Poplar Hill, Ontario, March 20, 1870, Dr. McLean received a public school education and then entered the Ontario Veterinary College. Following his graduation in 1892, he located at London, Ontario, later going to Crown Point, Ind.

During the Boer War, Dr. McLean entered the service of the British government and served in South Africa for a time. Later he was stationed at Lathrop, Mo., with the British Remount Division. Upon leaving this work, he located at La Porte, Ind.

Dr. McLean was a member of the Northern Indiana Veterinary Medical Association. He is survived by his widow (née Lena Grant), one daughter, one son, two sisters and one brother.

GEORGE E. BRISTOW

Dr. George E. Bristow, of DeKalb, Ill., died July 17, 1936, after an illness of but a few hours. He was born in Creston, Ill., June 12, 1860, and two years later his family moved to DeKalb. He was a graduate of the Chicago Veterinary College, class of 1891.

In 1904, Dr. Bristow was elected township tax assessor and he was engaged in this capacity at the time of his sudden death, having just completed the assessment of property for the 1936 taxes. He was said to be the best known citizen in his community.

FREDERICK C. BUSCHBOM

Dr. Frederick C. Buschbom, of Wilmette, Ill., died in the American Hospital, Chicago, July 27, 1936, at the age of 45 years. He was a graduate of the Kansas City Veterinary College, class of 1911, and had practiced at Dyersburg and Parkersburg, Iowa, before locating in Wilmette.

WILBUR R. MYERS

Dr. Wilbur R. Myers, of Oakland, Ill., died at his home, July 24, 1936, following a heart attack. He was born October 6, 1874, and was a graduate of the Indiana Veterinary College, class of 1907. He had practiced at Oakland for almost 30 years.

PERSONALS

BIRTHS

To Lt. and Mrs. Fred M. Sample, of Tyler, Tex., a daughter, Janice Marian, July 8, 1936.

To Dr. and Mrs. Tevis M. Goldhaft, of Vineland, N. J., a daughter, Linda Ann, June 15, 1936.

PERSONALS

Dr. A. A. McMurray (O. S. U. '24) has been transferred from Mercersburg to Norristown, Pa.

Dr. Ray Curry (K. S. C. '33) reports a change of address from Oklahoma City, Okla., to Selma, Kan.

Dr. Robert O. Biltz (U. P. '22), formerly of Camp Hill, Pa., has opened an office at Chestertown, Md.

Dr. H. L. Moser (O. S. U. '36) has taken over the practice of the late Dr. Frank T. Owens at Pontiac, Ill.

Dr. W. L. Hanson (McK. '08), formerly in practice at Aplington, Iowa, has located at Parkersburg, Iowa.

Dr. Kenneth H. Fraser (Mich. '35), is now associated with Dr. R. E. Bergman (Mich. '21), at Cassopolis, Mich.

Dr. E. A. Buxton (Chi. '90), of Vinton, Iowa, had his medicine-case and other equipment stolen from his car on May 2.

Dr. C. P. Brose (O. S. U. '27), has reported a change of address from Guilderland, N. Y., to Slingerland, N. Y., R. F. D. 1.

Dr. D. S. Hasson (Ont. '34), who has been assisting Dr. J. C. Flynn, of Kansas City, Mo., is now located at Mulberry, Kan.

Dr. A. A. Legner (Iowa '36), son of Dr. A. J. Legner (Chi. '09), of Leland, Ill., has opened an office at Mendota, same state.

Dr. John F. Ryff (Mich. '35), of Detroit, Mich., has taken over the practice of the late Dr. Levi L. Miller, at Caledonia, Mich.

Dr. W. T. Brinker (O. S. U. '30), of Miamisburg, Ohio, recently added a modern operating-table to the equipment of his hospital.

Dr. C. J. Dibbern (St. Jos. '19) reports a change of address from Pasadena to Los Angeles, Calif. Address: 3516 Harriman Ave.

Dr. George B. Jones (Ont. '94) of Sidell, Ill., has been ill with neuritis for the past few months, but is now on the road to recovery.

Dr. C. J. Kershaw (Mich. '30), of Plymouth, Mich., recently built a new veterinary hospital in the rear of his home at 9525 Wayne Road.

Dr. L. L. Willett (Gr. Rap. '11), formerly of Ravenna, Mich., has removed to Lake City, Mich., where he is engaged in general practice.

Dr. W. E. Roberts (U. P. '10), of Hazleton, Pa., will continue the practice which he conducted in partnership with the late Dr. A. C. Foos.

Dr. James M. Nelson (Iowa '08), of Sigourney, Iowa, narrowly escaped injury when his car was smashed in a collision in Sigourney, April 26.

Dr. N. H. Larson (Ind. '22), of Ossian, Iowa, has purchased and is remodeling a residence property, which he will occupy as his home and office.

Dr. T. J. Wagoner (G. R. V. C. '14), has removed from Greene, Iowa, to Vinton, same state, for general practice. He has purchased a home in Vinton.

Dr. F. L. Roach (Chi. '04), of Preston, Iowa, is reported as seeking the nomination for State Senator from Jackson County on the Republican ticket.

Dr. P. T. Engard (Ind. '13), of Marysville, Ohio, has been appointed a member of the State Board of Veterinary Examiners, by Governor Martin L. Davey.

Dr. W. C. Dendinger (St. Jos. '16), of Augusta, Me., was recently elected a member of the Board of Directors of the Rotary Club of that city, for the year 1936-37.

DR. THOMAS O'REILLY (K. S. C. '18), of Oklahoma City, Okla., accompanied by Mrs. O'Reilly, sailed for Europe on June 3, on the SS Washington. They will return to the United States in October.

DR. C. J. BUEHLER (K. C. V. C. '17), of Morton, Ill., narrowly escaped injury when his car was sideswiped by another car, four miles north of Delavan, the latter part of April. His car was badly damaged.

DR. CHARLES R. OMER (K. S. C. '29), formerly of Chestertown, Md., has accepted a position as field man with the Supplee-Wills-Jones Milk Company of Philadelphia, and is now stationed at Hagerstown, Md.

DR. CYRIL P. GOLDING (San. Fran. '11), of Kapaa, Kanai, T. H., is spending four months on the mainland of the U. S. A., with headquarters in San Jose, Calif. He will return to Hawaii in October.

DR. J. L. AXBY (Chi. '03), of Indianapolis, Ind., addressed the Washington Township Farm Bureau, at the Court House, Washington, Ind., on April 16. His subject was "The Control of Diseases of Live Stock."

DR. HERMAN DYKEMA (Mich. '28), of Muskegon, Mich., addressed a meeting of the Muskegon County Pomona Grange the latter part of April. His subject was "The Relations of the Veterinarian and the Farmer."

DR. ELVON S. DICKEY (K. C. V. C. '06), of Ottumwa, Iowa, recently retired from 30 years of active service with the U. S. Bureau of Animal Industry and has accepted a position with John Morrell & Company, of Ottumwa.

DR. ROBERT GRAHAM (Iowa '10), of the University of Illinois, addressed the Kiwanis Club of Bloomington, Ill., on April 20. His subject was "Relations Between Diseases of Food-Producing Animals and Public Health."

DR. E. C. WILLIAMSON (Ind. '15), of Montpelier, Ind., received serious burns when his clothing, which was saturated with sodium chlorate, used in spraying Canadian thistle on his farm, caught on fire, about two weeks ago.

DR. C. J. LANGE (Corn. '32) has resigned from the U. S. Bureau of Animal Industry, after several years of service, to accept a position as fellow in pathology at the Virginia Polytechnic Institute, Blacksburg, effective July 1.

DR. ERNEST HOGG (U. P. '14), of Wilkes-Barre, Pa., has been appointed veterinarian to the Kis-Lyn farm by the Board of Managers of the Luzerne County Industrial School for Boys. He succeeds the late Dr. F. J. McNeal.

DR. WILL WALKER (K. C. V. C. '14), of Golconda, Ill., was taken to the hospital recently as a result of wounds received in a gun fray with a burglar who entered his hardware store. The robber was killed, but it is believed that Dr. Walker will recover.

DR. R. L. ANDERES (K. S. C. '34), formerly a member of the staff of the North Shore Animal Hospital, Evanston, Ill., has joined the Jensen-Salsbery Laboratories, Inc., Kansas City, Mo., in charge of Clinical Medicine Research, a new service for practitioners.

DR. C. L. CRIDER (Iowa '14), of Elkader, Iowa, is president of the local school board and recently addressed 360 senior vocational students of 39 Iowa and Minnesota high schools, at Elgin, Iowa. His subject was "The Veterinary Field."

DR. WILLIAM H. IVENS (U. P. '10), of Philadelphia, Pa., was official veterinarian to the Atlantic City Horse Show in May. He was a member of the coaching party which drove from the Waldorf-Astoria Hotel, New York City, to Haddon Hall, Atlantic City, a distance of 134 miles, in ten hours and four minutes, a new mark for this trip.